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SECTION OF PEDIATRICS

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PRESIDING:
Dr. Edward S. O'Keefe, Lynn, Chairman.
Dr. Joseph Garland, Boston, Secretary.

CHAIRMAN O'KEEFE: The meeting will come to order. I will first appoint a committee to bring in a slate for the coming year for the Section. I appoint Dr. Richard M. Smith, Dr. Elmer W. Barron, and Dr. Charles L. MacGray. They will report later.

The first paper will be "Thyroid Disorders in Childhood" by Dr. Richard B. Cattell.

THYROID DISORDERS IN CHILDHOOD*

BY RICHARD B. CATTELL, M.D.†

NORMAL thyroid function is essential for proper mental and physical development throughout infancy and childhood. It, therefore, becomes very necessary to recognize any thyroid disorder or abnormal function so that proper treatment may be carried out, thus preventing later irremediable changes. Fortunately in this country there are few regions where goiter is endemic. In regions where it is endemic, disorders of the thyroid in childhood are common, and without proper prophylaxis and treatment they become quite serious and cause such conditions as dwarfism, idiocy and cretinism.

In this country, except for certain limited districts in the region around the Great Lakes and in Oregon, thyroid disorders in childhood may be considered as sporadic, that is to say, few babies or children are seen where a question of thyroid disease arises. On the other hand in a clinic where a large number of thyroid patients are seen over a period of years, a considerable number of children and young adults are encountered suffering from thyroid disease. Approximately one per cent of the patients presenting thyroid disorders coming to the Lahey Clinic are children under thirteen years of age. In many respects the conditions that these children present are similar to the diseases we see in adult patients. They differ, however, in very important ways. One sees in them certain arrests and changes in embryological development, delayed growth and mental retardation and fails to see conditions, the result of degenerative changes, that are present in long-standing goiter.

These disorders in infancy and childhood may be roughly divided into those that are developmental in origin, those that are functional and those that are of a neoplastic type. The following classification of them has been found to be satisfactory in dealing with these patients and this discussion will take up the diagnosis and treatment of the important ones mentioned. (Chart I.)

*Read before the Section of Pediatrics of the Massachusetts Medical Society, June 7, 1933.
The Lahey Clinic, Boston.

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CHART I

THYROID DISORDERS IN CHILDHOOD

1. Developmental Disorders
   a. Congenital absence of thyroid
   b. Lingual thyroid
   c. Aberrant thyroid
   d. Thyroglossal cysts and sinuses
2. Cretinism
   a. Congenital
   b. Sporadic
   c. Cachexia Strumipriva
   d. Hypothyroidism
3. Colloid or Adolescent Goiter (endemic goiter)
4. Hyperthyroidism
5. Inflammation
   a. Acute
   b. Chronic
6. Tumors
   a. Adenoma
   b. Aberrant thyroid
   c. Carcinoma

DEVELOPMENTAL DISORDERS

Congenital absence of the thyroid is very rare and is inconsistent with life. It may be discovered at autopsy in stillborn babies or fetuses showing other defects and is of little clinical interest. A consideration of the embryological origin of the thyroid is not pertinent to this discussion. It is sufficient to recall that the thyroid gland normally descends from its origin at the base of the tongue to its usual position in

the neck. Its descent may stop so that rarely the entire gland or portions of it may be found at any position along the normal course of descent. We have encountered three patients with a completely lingual thyroid present since childhood[1,2]. One was removed because of size and resulted in postoperative myxedema so that the administration of thyroid extract became necessary. This proved clinically that the lingual mass was all the thyroid tissue present in this patient. The other two patients were not operated upon. One died from leukemia and at autopsy it was clearly demonstrated that the lingual thyroid constituted all the thyroid, as there was none at the usual position in the neck. In spite of the considerable size which these swellings attain, at times filling the oral pharynx, they cause few symptoms of pressure or obstruction but they are at times the site of neoplastic lesions. They rarely require treatment but if it is considered necessary a partial resection should be attempted. In view of the reported cases it does not seem necessary to explore the usual position of the thyroid, as has been suggested, since there is little likelihood that thyroid tissue will be found. It is my belief that in the event some thyroid tissue is found along the thyroglossal tract it can then safely be assumed that this will be all that the patient possesses. I have been unable to find instances where separate and distinct portions of thyroid are found in the midline of the neck. This is not true, however, of genuine aberrant thyroids which may be multiple and are lateral in position[3]. These are definite tumor formations, and although they are also developmental in origin I should like to discuss them under the heading of tumors.

Thyroglossal cysts, sinuses and fistulae are not rare. It is commonly accepted that they are more frequent in infancy and childhood since they are developmental in origin. The age incidence, of course, depends to a considerable extent upon the relative number of children and adults seen. We[4] have treated more than eighty patients with this condition in the Clinic and of this number only fourteen per cent were seen in patients under twenty years of age. This is due to the fact that the majority of our patients are adults. Bailey[5] reviewed ninety-two cases collected from different London hospitals and found that the majority of these occurred in children. The diagnosis should not be difficult although in some children we have confused it with tuberculosis confined to the submental glands. There is usually a swelling present in the midline of the neck in the region of the hyoid bone. It rises on swallowing due to its close relation and attachment to the hyoid bone. A few cases have been reported with the cyst located in the lower portion of the neck in the midline but we have not encountered one in this position among our patients. A sinus may be present but this is usually the result of an incision into the cyst. The sinus usually drains intermittently. At times the cyst may be emptied into the pharynx by pressure over it, demonstrating a persistence of the thyroglossal tract. Rarely there is a direct communication from the pharynx to the anterior surface of the neck, or a true congenital fistula. The treatment is always surgical. Complete excision of the cyst or sinus together with removal of the central portion of the hyoid bone and a core of the muscles in a line up to the foramen cecum "en bloc" after the method of Sistrunk[6] is essential to effect a cure. (Illus. I.) We have

I. Excised thyroglossal cyst and tract. The radical operation has been done. Note the accompanying central portion of the hyoid bone.

operated upon three children under the age of ten years and seven others under the age of twenty years with but one recurrence. This patient was reoperated upon with relief. Carcinoma was associated with this cyst and I shall report it under the discussion of tumors.

CRETINISM

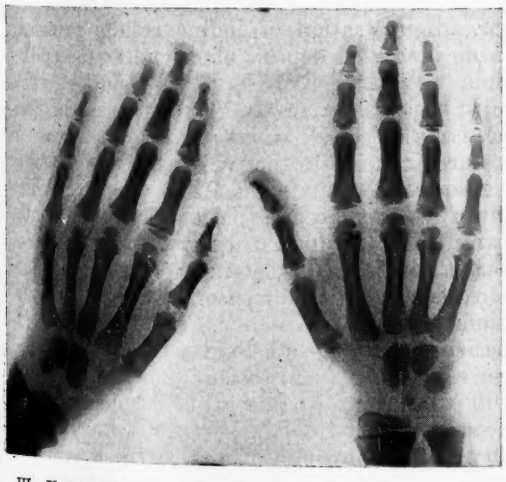
Cretinism is of little importance to us in this country but should be mentioned since occasional cases will be observed. The endemic form[7] occurs in goiter districts in children born of goitrous parents. It is associated with dwarfism, idiocy and typical physical characteristics, and may be with or without enlargement of the gland. These children are not born cretins but develop as such during early infancy. Treatment with thyroid extract will usually be of considerable benefit. Pregnant women with goiter, with or without evidences of hypothyroidism, should be given iodine during their pregnancy in order to avoid a goitrous child.

Proper goiter prophylaxis over a period of years by means of iodine as worked out by Marine and Kimball will effectively check it. The sporadic form occurs in non-goitrous districts with normal parents and is difficult to explain. These children do not usually have goiter and the condition is likely to develop undiscovered. The following history of such a patient illustrates this interesting condition[8].

CASE REPORT

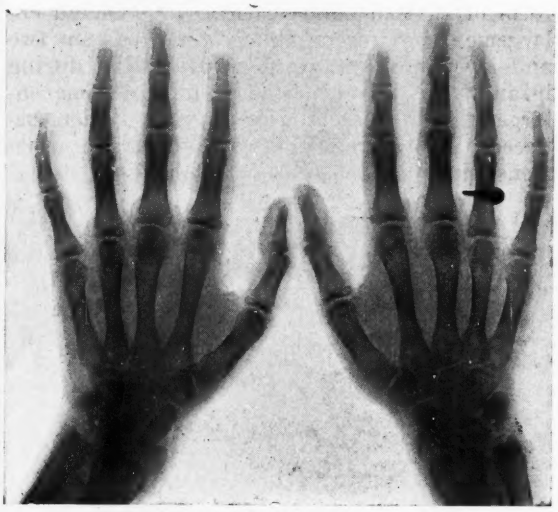
An American boy, eleven years of age, came to the Clinic because of a large tumor in his neck. A small lump was noted soon after birth with a gradual increase in size throughout his childhood. His skeletal development during early childhood was normal but had been delayed during the years of school age.

II. Photograph of a sporadic cretin, aged eleven. A very large goiter was present.



III. X-ray showing marked delay in appearance of ossification centers and in ossification of carpal bones. Compare with illustration IV.

His mental development was normal for his age. There was no familial history of goiter. He was forty-seven inches tall and weighed forty-six pounds. There was a large nodular soft tumor filling the right side of the neck and a smaller one involving the left lobe of the thyroid. (Illus. II.) X-rays of the carpal and tarsal bones showed a delayed ossification with a development equal to that of a normal boy of five years. (Illus. III.) There were no ossification centers in the patellae. The basal metabolism before operation was plus twelve and after operation was plus eight. The blood cholesterol was 212 milligrams. A subtotal thyroidectomy was done, leaving large sized remnants. In spite of the fact that the basal metabolic rates were found to be within normal limits, he is being treated with thyroid extract at present but sufficient time has not elapsed to demonstrate any change in ossification.



IV. X-ray of the hands of a girl, aged eleven, with exophthalmic goiter. Contrast the ossification of the carpal bones with those of the boy of the same age with cretinism in illustration III.

The true cachexia strumipriva or postoperative hypothyroidism is rare. The danger of removal of too much thyroid gland in children is well recognized at present. Hypothyroidism may follow the operative treatment of hyperthyroidism unless one has had considerable experience in the treatment of these cases[9]. Spontaneous myxedema or hypothyroidism is very rare although in certain children with retarded mental development it may be suspected. If proved by the clinical findings treatment with small amounts of thyroid extract will be of help.

COLLOID OR ADOLESCENT GOITER

Colloid goiter is not uncommon in non-goitrous regions and is the rule where goiter is endemic. This is the most frequent disorder of the thyroid in children and it becomes necessary for every physician dealing with them to be able to give proper advice concerning it. It is important to know what change has taken place in the gland as well as the etiological factors contributing to its production. The colloid enlargement may be the response of the thyroid to increased physiological demands but seems definitely related to an iodine deficiency. Colloid goiter shows a diffuse symmetrical enlargement of the gland produced chiefly by hypertrophy, that is increase in size of the thyroid cells, to a less extent by hyperplasia, that is increase in the number of the thyroid cells and

also by the deposition of colloid in the follicles. It is well recognized that the thyroid gland is a very labile and vascular organ and responds to such stimuli as generalized or local infection elsewhere in the body, menstruation, lactation, emotional stimuli as well as radical changes in diet. In children of goitrous parents there seems to be in addition a predisposition to this form of thyroid enlargement. This colloid enlargement may occur at any time between two and twenty years of age but rarely during infancy. It is very common to find some enlargement in the anterior cervical region between the ages of eight and the time when menstruation becomes fully established.

V. A prominent isthmus is evident in this girl, aged twelve. The thyroid is normal in size and consistency. A diagnosis of colloid goiter is frequently incorrectly made with these findings.

The diagnosis of this condition is not difficult although we find many children who have been said to have colloid goiter in whom we are unable to demonstrate any enlargement of the thyroid. This may be the result of an evident swelling in the region of the thyroid without being the gland itself. Particularly in young girls between the ages of ten and fourteen a transverse cervical fold of fat may simulate colloid goiter. This may be differentiated by picking up the prominence between the fingers demonstrating clearly that the swelling is limited to the subcutaneous tissues[10]. The other common mistake in diagnosis occurs in children of either sex who have very thin necks with a prominent larynx. In these children a normal thyroid isthmus may appear conspicuous, can readily be palpated and is frequently considered a colloid goiter. (Illus. V.) Measurements of the neck by means of a tape are of little value since it is very difficult to get measurements at the same position on the neck and because the normal body growth causes an appreciable increase in the size of the neck during childhood. The most important diagnostic aid is palpation of the thyroid. This is done after the method described by Dr. Lahey[11]. By this means one can demonstrate the soft enlargement of both superior poles, the lateral lobes and isthmus.

The treatment of colloid or adolescent goiter should be conservative. Of course the administration of small amounts of iodine, as recommended by Marine and Kimball in regions where goiter is common, is a very effective means of preventing it. As carried out under the direction of Klinger in Zurich where goiter is endemic the incidence in school children has been reduced from over ninety per cent to less than ten per cent. Because of the infrequency of the condition in our schools we do not believe iodine administered as such is to be recommended as a general practice. A well-balanced diet should provide sufficient iodine for normal thyroid function. The iodinization of both the water supply and table salt seems unnecessary although we do not believe it does any harm. These two simple methods of administering small quantities of iodine are of value in regions where goiter is endemic. It should be recognized that the administration of iodine results primarily in an increased deposit of colloid as the epithelium regresses or undergoes involution. This must necessarily result in an increase in firmness of the gland, if not an actual increase in its size at first. In the presence of colloid goiter we sometimes advise small amounts of iodine given weekly over a period of three months twice a year. This can be given as one drop of Lugol's solution or better in some more dilute form such as iodostarine or as a solution of sodium or potassium iodide. It has been our experience that once a colloid goiter has developed we cannot predict in what patients the goiter will disappear. Some will disappear spontaneously, others will disappear without treatment after possible related stress is removed, while still others remain unchanged in spite of any treatment. It seems best, as a general policy, to give small quantities of iodine and observe the patients every six months, but if the treatment "by neglect" is ever of value in thyroid disease it is in these patients and one is justified in giving no medication.

Rarely the colloid enlargement will continue and the thyroid will attain considerable size and surgical interference will be indicated either

because of superimposed hyperthyroidism, pressure or for cosmetic reasons.

It should be appreciated that when colloid goiter persists it is the first stage in the production of endemic or adenomatous goiter. Usually after twenty years of age nodules appear made up of adenomatous areas with areas of hyperinvolution followed by degenerative changes so common in this form of goiter. This condition is not of great importance in considering children other than in the prevention of its later occurrence. We have, however, operated upon two children (9 and 11 years of age) with this type of goiter, identical in appearance to the type seen in adults.

HYPERTHYROIDISM

Primary hyperthyroidism or exophthalmic goiter is not uncommon in children as previous to 1932 we had treated forty-five children with this disease fifteen years of age or less. (Chart II.) Hyperthyroidism may occur at any age

CHART II

AGE INCIDENCE IN EXOPHTHALMIC GOITER

4	2-6 years
1	9 years
1	10 years
3	11 years
2	12 years
6	13 years
17	14 years
11	15 years

after two years; the youngest that we have seen was two years and eleven months. (Illus. VI.) In its clinical and pathological manifestations it is quite similar to the disease seen in adults. All of the primary and most of the secondary signs and symptoms may be present. (Illus. VI, VII, VIII.) These children have exophthalmos, stare, widened lid slits and lid lag, activation, nervousness, irritability, a tremor that is frequently generalized, increased sweating and warm skin, tachycardia, palpitation and loss of strength as well as typical hyperplastic thyroid glands. The basal metabolism is regularly elevated. The diagnosis is usually easy if one thinks of the possibility of hyperthyroidism occurring in children. In cases where the diagnosis does not appear certain, they should be carefully watched with frequent examinations and without iodine medication. In patients in whom the diagnosis is obscure, iodine may mask the disease in causing temporary improvement. The basal metabolism test is not always reliable in children and cannot be de-

VI. A girl, aged two years and eleven months, with exophthalmic goiter. Note the outline of the hyperplastic gland with the enlarged superior poles.

VII. Exophthalmic goiter in a girl, aged four and one-half years. There was marked exophthalmos. The gland was firm and one and one-half times its normal size.

VIII. Exophthalmic goiter in a girl, aged nine. All of the typical manifestations of the disease are present.

pended upon to the same extent as in adults, because of technical difficulties in getting the child to breathe through the mouthpiece without leakage. If a direct means for determining the basal metabolic rate were at hand, it would of course be consistently elevated. In the absence of an elevated metabolism in these children we do not make the diagnosis of hyperthyroidism.

With the diagnosis established we believe all of these children should be operated upon. An operation correctly done will relieve the patient quickly and will permit the child to go on with normal development. There is a rather common feeling that they should not be operated upon because they are so young and because they might be carried along with medical treatment. If unoperated and treated medically with complete rest, small amounts of iodine and sedatives, the condition will not be cured. We do not believe x-ray therapy should be employed since it is too difficult to gauge with sufficient accuracy to get relief and not get hypothyroidism. It cannot be depended upon to the same extent as the operative treatment.

The operative management of these children is a somewhat different problem from the treatment of adults. They are kept on a preoperative treatment until they show a satisfactory weight gain. This is frequently a longer period than is necessary in adults. We believe it best to divide the operation into two stages in most cases since no one's experience is sufficient in this group to know how well the individual child will tolerate surgery[12]. The stages are done with an interval of six weeks during which time they are given five drops or less of Lugol's solution daily. The operation is identical except for the amount of tissue to be left as a remnant. While it is impossible to state the amount to be left in any numerical fraction, it is important to leave relatively larger remnants than one leaves in adult patients. The results of operation are very satisfactory. We have had but one child operated upon who developed a recurrence of the hyperthyroidism and required a second operation. In none was growth interfered with and it has not been necessary to give thyroid extract to any of these patients postoperatively. There was no instance of postoperative parathyroid tetany. There was one postoperative death occurring in 1925 in a boy eleven years of age who died in a postoperative thyroid storm after a first stage hemithyroidectomy.

INFLAMMATION

Thyroiditis is very rare in children. The thyroid seems particularly resistant to serious infection although it is very apt to show hypertrophy as a secondary manifestation. Mild degrees of inflammation with local tenderness may at times be demonstrated associated with tonsillitis, cervical adenitis and generalized infections. These do not require treatment. Chronic thyroiditis with local symptoms of pressure are not encountered. We have seen only two cases of tuberculosis and three of syphilis that were primary in the thyroid and none of these were in children.

TUMORS

The only benign tumor of the thyroid is the discrete adenoma. It is quite probable that these develop in fetal rests and are present since the development of the fetal thyroid. If this were the case it would be logical to assume that a certain number could be discovered during infancy and childhood but this has not been true. It is a rare finding in children and in our experience has been encountered only three times. In these patients the findings were identical with those found in adults both as to the history and examination of the thyroid. (Illus. IX.) In all three children the adenoma was discrete, with the remainder of the gland of normal consistency. The treatment should be surgical removal at any age. In no instance under any other treatment, either in adults or children, have we seen these tumors actually disappear. They may of course descend into the substernal space and no longer be palpable but in this position can be demonstrated by x-ray. Since they are the source of practically all carcinoma of the thyroid they must all be removed if the incidence of malignancy is to be lessened. Their removal in no wise is a detriment to normal growth as the thyroid itself is left intact.

IX. A discrete fetal adenoma is shown in a child, aged twelve.

Lateral aberrant thyroid tumors are of considerable interest both because of clinical and pathological inconsistencies. Embryologically they develop from the lateral pharyngeal

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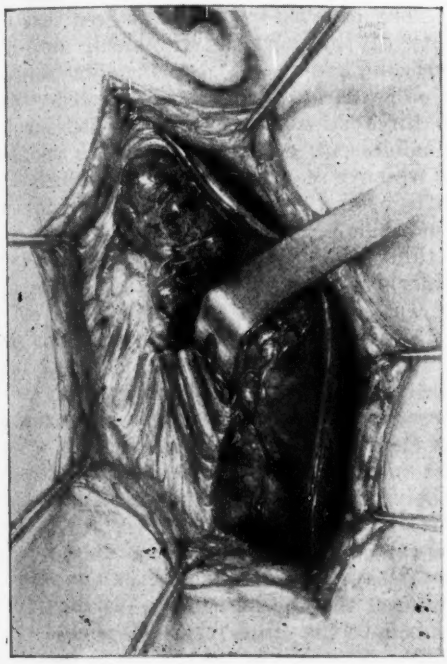
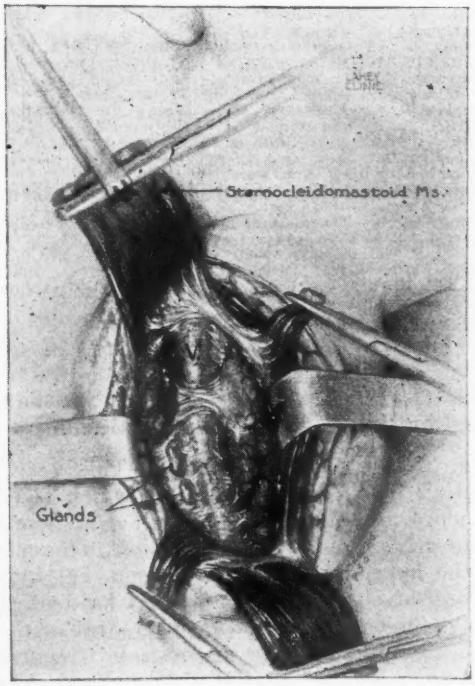
pouches that normally do not take part in the formation of the thyroid gland. This small group of cases has been little understood and was reported by the writer recently[2]. They may occur at any age although but two occurred in children (eleven and thirteen years of age). They are found either as discrete tumors or as multiple chain-like tumors simulating cervical adenopathy. The diagnosis is always uncertain without biopsy although they may be suspected when single or multiple firm tumors are found



X. A drawing made at operation of a discrete lateral aberrant thyroid nodule in a girl aged thirteen. The superior pole of the right lobe is involved with papillary adenocystoma, although distinct from the aberrant nodule. The lymph glands were not involved.

on one side of the neck, present for a considerable time without fluctuation in size but with a gradual and progressive growth. They are always present without symptoms and with the thyroid gland itself usually normal on palpation. The microscopic picture is characteristic, showing the structure of a papillary cyst adenoma not unlike that observed in certain ovarian cysts and breast tumors. At times they show no typical thyroid structure as evidenced by follicles or colloid. They may show any degree of differentiation and as in the case of thyroid carcinoma it is difficult to determine the degree of malignancy in the sections. The treatment is local excision of the discrete tumors and block dissection of the neck when they are multiple. (Illus. X-XI.) They do not metastasize but spread by local extension and do not tend to recur. The results of surgical excision combined with postoperative radiation are good.

We have had three cases of carcinoma of thyroid origin in children, which I have recently reported[13]. They occurred at the ages of six, eleven, and thirteen years. The first was an adenocarcinoma (grade one) in a thyroglossal cyst occurring in a boy of six years. A radical operation was done and the malignancy discovered in the operative specimen. A few days later a second radical excision was done to be sure all malignant tissue was removed. The cyst then recurred and a third radical excision was done. He has remained free from recurrence for eighteen months. The second case, a girl of eleven years, was an adenocarcinoma in lateral aberrant thyroid tissue with local invasive characteristics. This was removed by block dissection of the neck combined with complete hemithyroidectomy on that side. With postoperative x-ray therapy she has remained free from recurrence for over two years. The third patient was a boy of thirteen years who had a small cell carcinoma involving all of the right lobe of the thyroid producing a marked degree



XI. Lateral aberrant thyroid tumor in a girl, aged eleven, with direct invasion of the cervical structures. This was an adenocarcinoma. A radical neck dissection was done followed by x-ray therapy and the patient has been free from recurrence for two and one-half years.

of tracheal stenosis. A large amount of the malignant tissue was excised and tracheotomy done. With x-ray treatment all palpable and x-ray evidence of the tumor disappeared and he appeared well for nine months; after this it recurred rapidly and caused his death in thirteen months. This last patient shows the same clinical course as an adult with the same type of cancer.

SUMMARY

In reviewing this discussion it can be seen that disorders of the thyroid in children are not unusual. Advice will be frequently sought of the physician regarding the presence or absence of colloid goiter and the treatment for it. Small quantities of iodine given for a short period semiannually are sometimes of benefit in colloid goiter and do no harm. Hyperthyroidism will be encountered with the same clinical characteristics as in adults and subtotal thyroidectomy should be done, the results of which are very satisfactory. The other disorders are more rarely encountered and although sporadic must be recognized and properly treated if these children are to develop normally.

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Discussion

CHAIRMAN O'KEEFE: This paper will be discussed by Dr. Fritz B. Talbot, of Boston.

DR. TALBOT: *Mr. Chairman*—This very interesting paper opens a subject which is coming more and more to the front and deserves considerable thought and discussion. One might get the impression from the paper that hyperthyroidism is the most important kind of thyroid pathology that we have to deal with in this community. Those impressions frequently arise from the fact that statistics of specialized clinics do not represent a cross-section of what is happening in the community. It would appear from the paper as if goiters were common and that hypothyroidism was rare in this community, so I should like to read a few figures of the incidence of thyroid diseases in the children's metabolism clinic of the Massachusetts General Hospital and in my own private practice.

The age limit of these patients is about puberty. There was one congenital cretin. A congenital cretin being a child who has all the clinical signs of cretinism at birth, and there were 68 sporadic cretins. I believe that a sporadic cretin is also congenital, but the clinical signs do not appear until about the third month. There were twelve cases of juvenile myxedema. I mean by that myxedema which appears for the first time later in childhood, and there were fifteen cases of hypothyroidism without any signs of myxedema.

There were six cases of exophthalmic goiter in children under ten years of age. Exophthalmic goiter or toxic goiter is very rare in very young children. The incidence increases with age. The number that will be seen in a community of this sort is very small until after puberty. There were two congenital goiters, a pair of twins born with large thyroids. There were three adenomatous goiters and innumerable adolescent goiters; but of the adolescent cases we have seen we have obtained metabolism on fifteen, and the metabolism in all instances was normal.

I think the most important question when considering goiter and operation is the diagnosis, and I am sorry that the speaker did not make clearer how the differentiation was made of colloid and toxic goiters. Since operation is contraindicated in the former, which may be confused with the latter, every precaution should be taken to be sure of the diagnosis.

The speaker did not have time to discuss the metabolism which is mentioned in his original paper. I am going to take a moment to speak of the value of basal metabolism. In the original paper it says that it is so difficult to obtain it in children that he has omitted having it done, but I believe his clinic places dependence on it in adults.

Basal metabolism cannot be obtained by means of the portable apparatus in children under eight years, excepting those rare cases which are trained for the test. It can be obtained in the cot chamber at any age and is continually being done in the children's metabolism clinic at the Massachusetts General Hospital.

The question of diagnosis, as to whether we are dealing with a colloid goiter or a toxic goiter requiring operation, is a very important one because in making that decision comes the quarrel between those who prefer medical treatment for children and those who say that surgical treatment is necessary. My cases, which I have called toxic goiter, were all treated medically. Three of the cases I have been able to follow over a long period of time, one of them over eight or nine years, and so far as I can see the treatment has been completely successful. That would lead one to question whether, in all cases of toxic goiter, operations are necessary. I should like to repeat what I wrote some years ago, that I consider operations necessary in toxic goiter when the goiter is large enough to cause obstruction to the breathing and when

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medical treatment has not been successful and shows no promise of success.

In regard to adenomatous goiter, I am not sure that I am talking about the same condition as Doctor Cattell, but I have in my experience seen three cases all of which received iodine with an increase in the size of the goiter and in symptoms. It seems to me that in these individuals iodine is distinctly contraindicated.

The surgical aspect of this subject has been so well presented that I will not say anything about it. In regard to medical treatment, I think that the most important factor is to eliminate stress. I have a case of goiter with staring eyes which came to me four years ago and at that time had a metabolism of plus 60 per cent. With medical treatment about a year ago the metabolism came down very close to normal. The pulse at the beginning was 100 to 120. The child on the first visit hopped all over the room. With medical treatment, the metabolism, as I said, came down to normal limits. The pulse came to the vicinity of 65. The tumor swelling has become smaller and smaller so that the last time I saw her the tumor was not recognizable.

I think the paper opened a subject which is very important. Disease of the thyroid is much more common in children than was earlier believed. I thought ten years ago that I knew nearly all that there was to be known about the thyroid gland and about hypothyroidism and hyperthyroidism. The changes in the knowledge of the relationship of all glands has been so great in the last few months and years that nobody can predict how much we will have to modify our present opinions. No one can tell where this fascinating road is leading.

CHAIRMAN O'KEEFE: This paper will be open to general discussion now. Is there anyone who desires to discuss it? If so, I will ask him to announce his name and address in a loud, clear voice, and I think it will help if those who are going to discuss the papers will come to the front of the hall. The discussion will be limited to two minutes. If there is no discussion of this paper, does Doctor Cattell wish to say anything in closing?

DR. CATTELL: I presented this subject this morning as the experience in a thyroid clinic. As intimated in the paper, our figures are quite different from those of most hospitals, particularly those where the greater proportion of the cases are children. This explains the difference in the incidence of the thyroid diseases observed by Dr. Talbot. He has called attention to the relative frequent finding of hypothyroidism. Dr. Lewis M. Hurxthal has called attention to the value of blood cholesterol determination in adult patients with true myxedema and it is equally important in determining whether thyroid extract would be of benefit in children. He showed that if the blood cholesterol were increased, accompanied by a low basal metabolic rate, thyroid extract would be beneficial.

As pointed out in my paper exophthalmic goiter was a fairly frequent finding, since forty-five cases were encountered who were fifteen years of age or under. All of the children that we have observed with hyperthyroidism were true exophthalmic goiters and we have not observed the toxic adenomatous goiter mentioned by Dr. Talbot. There seems to be a general feeling throughout this country that the value of the metabolism test is considerably overrated. At this clinic, however, we believe it to be of great value and utilize it in all cases. A diagnosis of exophthalmic goiter in a child should not be made unless an increased oxygen consumption, that is to say an increased metabolic rate, is shown. We believe that all of these children should be operated upon in order to get satisfactory results.

There is considerable literature that deals with the use of iodine in colloid goiter. If you give iodine to patients with that type of gland, the gland will usually grow larger in the beginning. This results because of the increased deposit of the iodine-bearing colloid and it, no doubt, explains why iodine is of so little value once a colloid goiter has developed. On the other hand, it is of great value in the prevention of this type of goiter in children.

CHAIRMAN O'KEEFE: Is there any further discussion on this paper? If not, we will proceed to the next, entitled "What the Pediatrician Can Do for Dental Cripples," by Dr. Cleophas P. Bonin, Boston.

WHAT THE PEDIATRICIAN CAN DO FOR DENTAL CRIPPLES*

BY CLEOPHAS P. BONIN, D.M.D.†

THE dental profession always welcomes its medical friends to come before it because it invariably leads to a better understanding and closer coöperation between the two professions. In turn, those members of my profession who have the advancement of their work at heart always welcome an invitation to appear before their medical friends in their endeavor to further strengthen these bonds of mutual understanding and interdependence.

I therefore sincerely hope that the time set aside this morning for the discussion of ortho-

*Read at the Annual Meeting of the Massachusetts Medical Society before the Section of Pediatrics, at Boston, June 7, 1933.

†Bonin—Member of Orthodontia Teaching Staff, Harvard Dental School. For record and address of author see "This Week's Issue," page 920.

dontia will prove to be not only of benefit to you pediatricians, but, by the interdiscussion which I trust will follow, will also bring about another forward step in orthodontia itself.

The orthodontist and the pediatrician have one great and common bond, the welfare of growing children. When you gentlemen realize that the mouth is the seat of one of the five great senses, taste, that the mouth is the mould for every spoken word, and that it is in the mouth that all voluntary digestion takes place, I am sure you will agree with me that a normal mouth means much to the child's future.

In the short time allotted to me, all I can attempt to do is to tell you something about the orthodontist's field of work, stressing particularly our desire to eradicate the causes of malocclusion and thus prevent, so far as possible, these mouth deformities. I am most hopeful that my little offering will, as I already pointed out, lead to a discussion among you pediatricians with the rhinologists and the orthodontists present, which will bring about a further prevention of these deformities which have such a far-reaching, pernicious influence on the child's future.

SLIDE 1

Here we see the casts of the temporary dental arches of two children at about five and one half years of age. One of these children is going to have a normal permanent dental appara-

(Text-book.)
SLIDE 1.

tus while the other set of casts indicate that that child, all things being equal, will have a deformity of the permanent dentition serious enough to interfere with function.

Inasmuch as you pediatricians are the custodians of the health of children of this age, it may be helpful to you, if of course you do not already know which child is going to have a normal mouth with normal dental arches and normal development of the internal structures of the face, and which child is going to have an abnormal mouth and collapsed dental arches accompanied by pinched and cramped nasal spaces.

Note that the casts on the right show a spacing between all the anterior teeth while those on the other casts are all in contact—the arches on the right are normal for that age, while those on the left are abnormal. There are certain abnormal forces at work here which are interfering with the development of the dental arches and bones of the face of that child. This child is a chronic mouth breather and is going to have a mouth deformity.

Before a group of pediatricians, it is, of course, not necessary to dwell long upon the evil effects of mouth breathing, for the treatment of perverted breathing during childhood is within your field of work. I might point out, however, that chronic mouth breathing, brought about usually by enlarged adenoids and diseased tonsils, is a common cause of mouth deformity. As you so well know, when the nasopharynx is blocked by adenoids or diseased tonsils, or both, the child, in breathing, is forced to use the relief valve—the mouth. The continued open mouth and sagging lower jaw upset the equilibrium of the forces of the facial muscles. The constant but slight inward force thus brought about by muscles of the cheek and lip on the delicate developing bones of the face and the dental arches, causes a deformity to both. All of this, of course, is not new to you, but I do wish to emphasize that the early correction of mouth breathing is preventive orthodontia of the highest order. I beg of you, therefore, to see to it that the upper respiratory tract is cleared of all obstructions early in the child's life. However, in those cases of mouth breathing which have come to you after the child has become a dental cripple, then the orthodontist stands ready to coöperate with you for the benefit of the child's future.

SLIDE 2

This case was treated by Dr. Ketcham. The rhinologist, Dr. Carmody, made accurate measurements of the posterior nares and found that those spaces were half the normal size when the treatment for straightening was started. In approximately a year, or when the active work of straightening and expanding the dental arches was completed, the second postnasal examination revealed that the spaces had increased from two-thirds to three-quarters of normal. The final examination was taken four years later.

SLIDE 2.

Please note how greatly the posterior nares have now increased. The first benefit came as a direct result of orthodontic treatment while the last was all gained from an established normal function. Far greater would have been the benefits had this patient been treated at the first indication of the deformity which undoubtedly manifested itself in the temporary dentition. This shows the splendid coöperation of the rhinologist and the orthodontist.

New England

Slide 3

To show you how important normal function is for the proper development of the jaws and the whole structure of the head, I now wish to show you a piece of research conducted some twenty years ago by the present Professor of Orthodontia at the Harvard Dental School, Dr. Lawrence W. Baker. He took several groups

SLIDE 3.

of animals and interfered with the occlusion on one side of each animal so that function on that side was eliminated. Note how the whole head of the dog shown in this slide is twisted. The working side is well developed while the growth of the interfered side has been inhibited. Notice the difference in the development of the two zygomatic spaces.

Slide 4

Here is the upper aspect of the same skull. Note that the deformity has extended throughout the entire skull. Every bone and every su-

SLIDE 4.

ture has been deformed. This experiment, gentlemen, clearly shows how tremendously important function is to the developing child, beginning at birth.

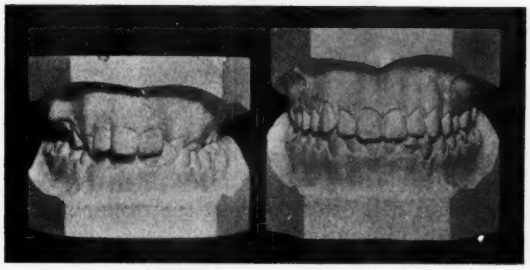
Slide 5

The casts on the left are those of a boy fourteen years of age. His temporary dental arches could have shown no spacing. In fact, in all probability they were constricted and gave early evidence that a dental cripple would result. Enlarged adenoids and diseased tonsils were undoubtedly the primary cause of this condition, although malnutrition was possibly a contributing factor, and lack of proper function for years no doubt did a great deal in further increasing the malocclusion. We prefer to prevent such conditions rather than to treat them. This boy continued to have colds long after his adenoids and tonsils had been removed, but the latest report from him was that he has had no colds for the last two years because his posterior nares had been enlarged, he no longer is a mouth breather, and bone growth has been greatly increased by normal function.

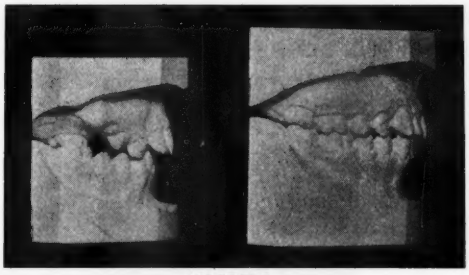
SLIDE 5.

Slides 6—7—8—9

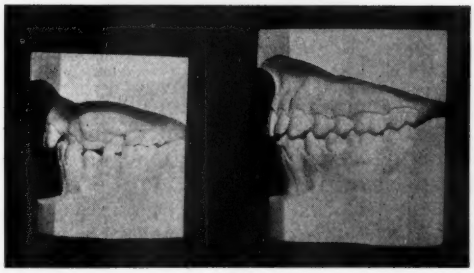
These show the casts of a little girl ten years old. Please note how the whole left side of the maxilla is in lingual occlusion, that is, it fits inside the mandible. Notice how the upper jaw has been distorted. The same causes enumer-



SLIDE 6.

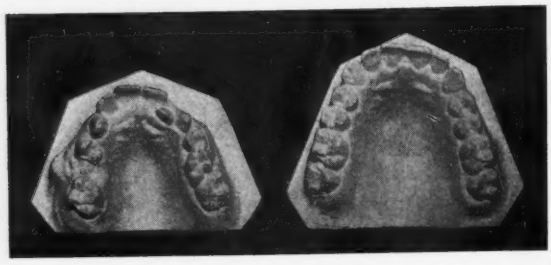


SLIDE 7.



SLIDE 8.

ated in the previous case were undoubtedly responsible for this condition. About all the various applied forces known in orthodontia were employed to treat this dental cripple.



SLIDE 9.

Slide 10

Let us now examine the skull at birth. Note the lack of vertical development from the lower border of the orbit to the lower margin of the mandible. Compare with the lower border of

(After Turner.)
SLIDE 10.

the orbit to the top of the head. What is to increase this vertical development so that a better proportion will exist between the upper and the lower half of the head?

Slide 11

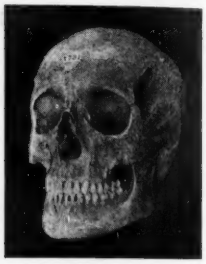
The next slide shows the skull of a child of about six years. An immense amount of development has taken place between the orbit and

(After Angle.)
SLIDE 11.

the lower border of the mandible. The cause is apparent—the fully erupted and normally functioning deciduous dentition and the developing permanent teeth.

Slide 12

What a beautiful result of normal influence is seen in this adult skull. Let us pause a moment to study the diagnostic landmarks in orthodontia, the primary one being the relation of the first molars, next the canines, and then the relation of the balance of the teeth, one to the other, and also the great importance of the relation of the mandible to the maxilla.

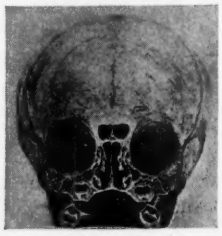


(Harvard Dental School Museum.)
SLIDE 12.

Time does not permit me to discuss the various types of malocclusion. However, I must take time to briefly describe the three basic forms. Class I—the relation of the jaws are normal with a protrusion or retrusion of the anterior teeth, and a contraction of both jaws with crowded and rotated teeth. Class II—the mandible is displaced backwards to normal. Here we may have a maxillary protrusion or retrusion. Class III—a forward relation of the mandible to normal. I shall call your attention to these various types as cases are thrown on the screen.

Slide 13

The next slide shows a cross section of the skull at birth. Here you will see the small antrum spaces. All the crowns of the deciduous teeth are formed at this stage of development including those of the first permanent molars, commonly known as the six-year molars. More



(After Cryer.)
SLIDE 13.

than that, gentlemen, the buds of all the permanent teeth have been laid. You therefore can well appreciate how important a well-balanced diet is during pregnancy and that from birth the new-born be breast fed so that he will have to work for his meals and immediately bring into action the muscles of mastication and all others pulling on the bones to which they are attached, thus stimulating bone growth.

Slide 14

This is a cross section of the adult skull. The vertical growth has been tremendous. The antrum has so increased from its size at birth that the dimension from the orbit to the lower

(After Cryer.)
SLIDE 14.

margin of the mandible of the skull at birth could now be put inside of the antrum. In fact, the whole maxilla has developed so that it is now able to support the greater dimension of the permanent teeth.

SLIDE 15

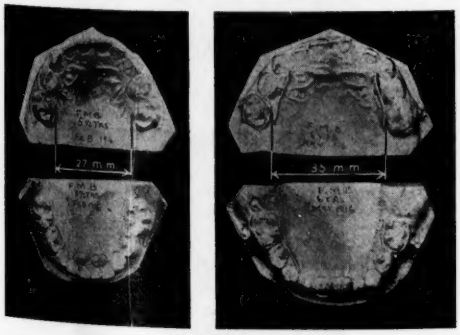
This case is from the office of a pioneer of early treatment, Dr. Bogue of New York. He informed the mother of this child that he advocated the immediate broadening of her temporary dental arches because he could foresee

SLIDE 15.

trouble. The mother was quite indignant when thus informed as she felt that her child had such pretty, regular teeth. You will notice that the teeth are all in contact, indicating that some evil cause is at work. Dr. Bogue insisted, however, that the mother at least take the child to a rhinologist to have her enlarged adenoids and diseased tonsils removed.

SLIDE 16

Six months later, Dr. Bogue's telephone rang and it was this same mother, quite upset because

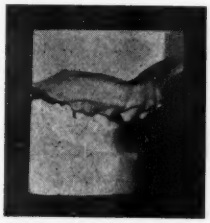


SLIDE 16.

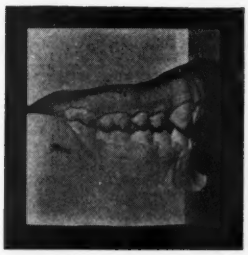
the first permanent lower teeth were erupting lingually, out of line of occlusion. The adenoids and tonsils were operated upon and the cause removed. Dr. Bogue then placed very simple appliances on the child's teeth and six months later had broadened the arches eight millimeters and thus prevented what might have become a very complicated orthodontic case.

I will now present a few cases which show what has been done for dental cripples at an early age:

CASE 1. Aged 3 years, 9 months. The collapse of the mandible is due to the sucking of four fingers as well as to enlarged adenoids and diseased tonsils. Note the backward position of the mandible due to the narrowing of the maxilla. The collapse and the backward displacement of the mandible have caused the maxilla to "hang in the air", so to speak.

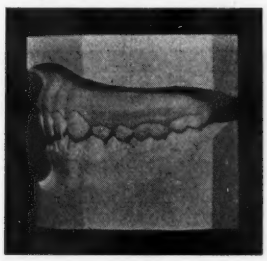


CASE 1, FIG. 1A.



CASE 1, FIG. 1B.

CASE 1, FIG. 2A.



CASE 1, FIG. 2B.

In figures 1-A and 1-B a period of eleven years has elapsed. The normal functional forces which have been established have done much to further the proper development of the bones of the face and its internal structures. Figures 1-A and 2-A show the right and left sides before treatment and figures 1-B and 2-B after treatment. Figures 3 and 4 show the occlusal surfaces.

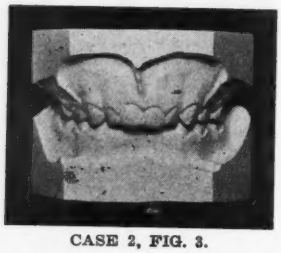
FIG. 3.

FIG. 4.

CASE 2. Aged 4 years. This case is presented to show how early in life many dental deformities are manifested, which in all probability, have existed for a year or longer. This case shows a marked displacement of the mandible and very constricted

CASE 2, FIG. 1.

CASE 2, FIG. 2.



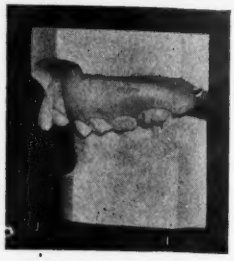
CASE 2, FIG. 3.

arches. While this child is a thumb sucker, it is my firm belief that these conditions are inherited rather than due to environment.

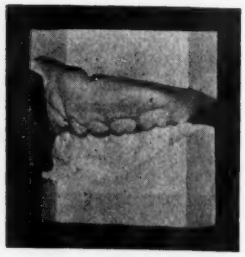
Treatment of this case is to be started in the fall.

Figures 1 and 2 show the right and left sides. Figure 3 shows the front view.

CASE 3. Aged 8 years. Figures 1-A and 2-A show a backward displacement of the mandible on both sides with protruding maxillary incisors. In figure 3-A you will notice a frenum between the superior



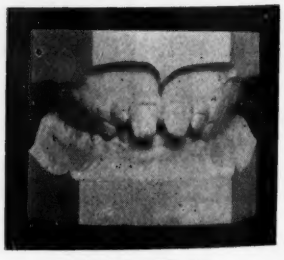
CASE 3, FIG. 1A.



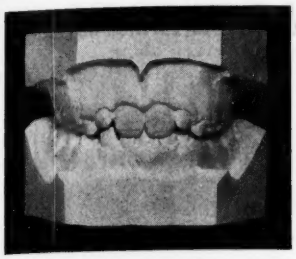
CASE 3, FIG. 1B.

CASE 3, FIG. 2A.

CASE 3, FIG. 2B.

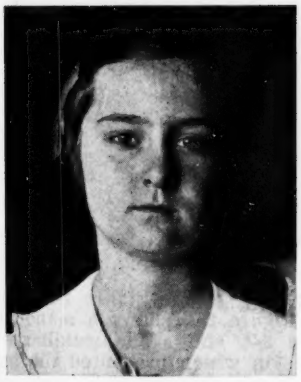


CASE 3, FIG. 3A.



CASE 3, FIG. 3B.

CASE 3, FIG. 4A.



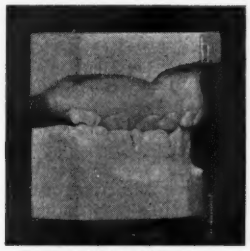
CASE 3, FIG. 4B.

CASE 3, FIG. 5A.

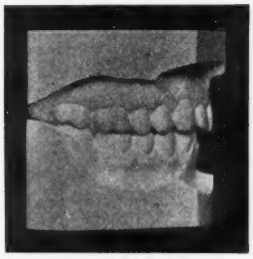
incisors. In figures 1-B and 2-B you will note that the dental arches have assumed their normal anatomical relationship and in figures 4-B and 5-B you will note how the treatment has improved the facial lines.

CASE 3, FIG. 5B.

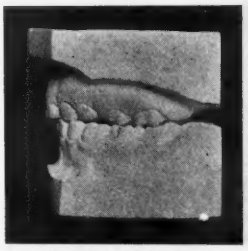
CASE 4. Aged 6 years. Figures 1-A, 2-A, and 3-A show conditions before treatment. In figures 1-B, 2-B and 3-B we see the changes eight years later. In figure 2-A the mandible was misplaced back-



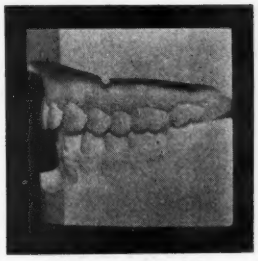
CASE 4, FIG. 1A.



CASE 4, FIG. 1B.

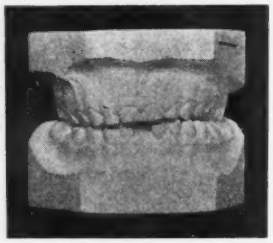


CASE 4, FIG. 2A.

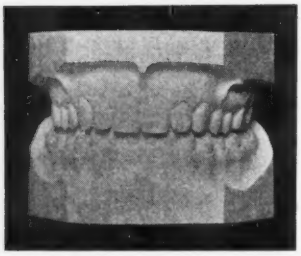


CASE 4, FIG. 2B.

ward. Figure 3-A shows the maxilla in lingual occlusion and also warped. Note how all the forces of malocclusion have been overcome and what influences these corrections have had on the proper development of the face and its internal structures.



CASE 4, FIG. 3A.

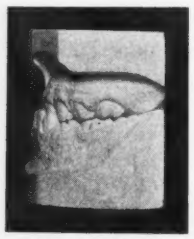


CASE 4, FIG. 3B.

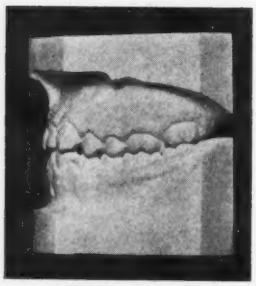
CASE 5. Aged 6 years. Figures 1-A, 2-A and 3-A show a mandibular prognathism and the malocclusal forces are inhibiting the normal development of the maxilla.

Figures 1-B, 2-B and 3-B show the corrected conditions. The mandible has been pushed backward and the establishment of normal forces had a powerful influence to further develop normal bone growth.

Nine years have elapsed between figures A and B.

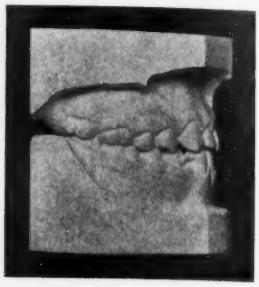


CASE 5, FIG. 1A.

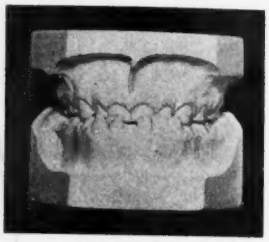


CASE 5, FIG. 1B.

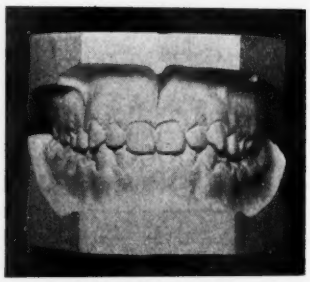
CASE 5, FIG. 2A.



CASE 5, FIG. 2B.

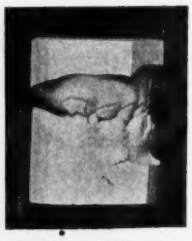


CASE 5, FIG. 3A.



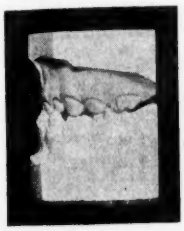
CASE 5, FIG. 3B.

CASE 6. Aged 3 years. Figure 1-A shows normal right side. Figure 2-A shows a mandibular protrusion. Figure 3-A shows the maxillary incisors in lingual occlusion which prevented the maxilla from developing. It was necessary to work only on the

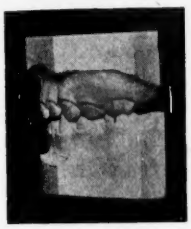


CASE 6, FIG. 1A.

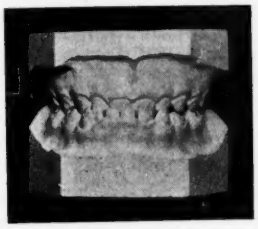
CASE 6, FIG. 1B.



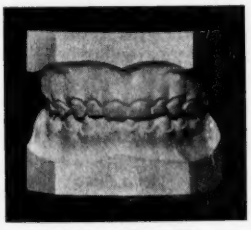
CASE 6, FIG. 2A.



CASE 6, FIG. 2B.



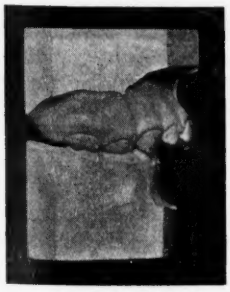
CASE 6, FIG. 3A.



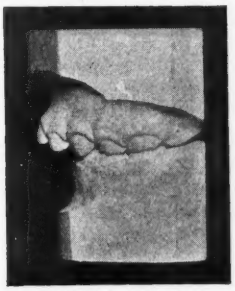
CASE 6, FIG. 3B.

maxilla to establish normal occlusion. Nature was then given a chance to develop normally from then on. This child's jaws have been made to function normally which is all that can be done now. It required but three months' treatment to make the corrections as shown in figures 1-B, 2-B and 3-B.

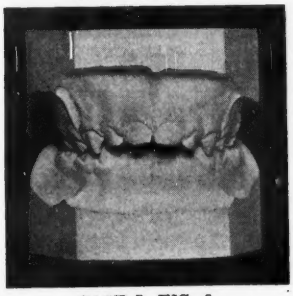
CASE 7. Aged 4 years. This case presented such a marked maxillary protrusion that it was impossible for this child to close his lips. This condition was accompanied by a tongue habit as shown in figures 1, 2, and 3.



CASE 7, FIG. 1.

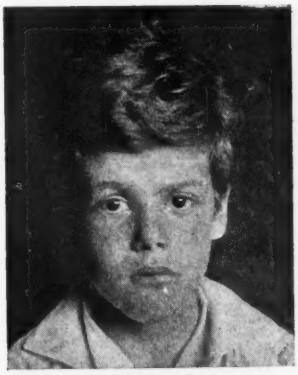


CASE 7, FIG. 2.



CASE 7, FIG. 3.

CASE 7, FIG. 4.

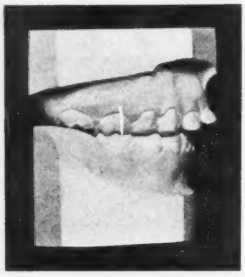


CASE 7, FIG. 5.

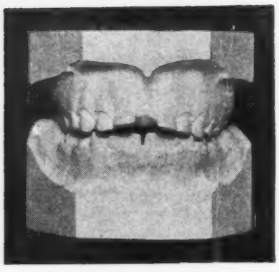
CASE 7, FIG. 6.

Figure 4 shows the boy before treatment. Figures 5 and 6 show him as he is today. Note how well he is able to keep his lips together without conscious effort. In his present condition, normal breathing having been established, his lips and jaws have been given a chance to develop normally.

CASE 8. 7 years old. This case is shown at this time because the pediatrician who cared for this child told the mother when the child was but three weeks old that considerable trouble could be expected with the child's jaws. This presents as difficult



CASE 8, FIG. 1.

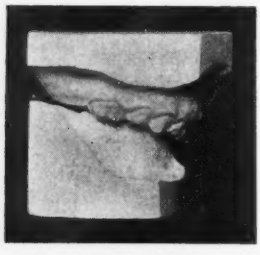


CASE 8, FIG. 2.

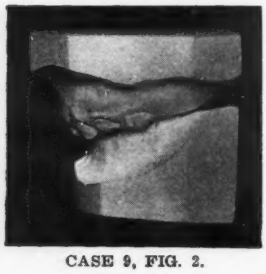
a habit to overcome as any orthodontist is ever called upon to break, namely, a tongue habit This condition is complicated by an extremely large frenum, as shown in figure 2, and congenitally missing permanent lateral incisor, as revealed by an x-ray.

This case is to be treated in the fall.

CASE 9. Aged 6 years. For the child's sake, I sincerely hope that this boy was given orthodontic

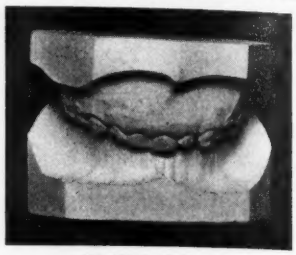


CASE 9, FIG. 1.



CASE 9, FIG. 2.

treatment for he was one of the worst dental cripples I have ever examined. Figures 1, 2, and 3 show that the mandible is completely in lingual occlusion with an excessive overbite of the maxillary



CASE 9, FIG. 3.

incisors. It is hard to understand how this child could masticate at all.

CONCLUSION

We orthodontists at times learn that pediatricians fear that orthodontic treatment undertaken at a very early age will cause a child's nervous system to become upset. This may be true if treatment is undertaken by unskilled hands but if properly treated such dangers do not exist. I believe, furthermore, that the older a child becomes, the more highly organized the nervous system becomes and consequently the dental deformities are far more complicated and therefore there is greater danger of upsetting the nervous system.

The conditions shown by the casts in this paper cannot help but influence the future of these dental cripples. These deformities go deeper than mere facial disfigurements and are so closely allied to the highest nerve centers that their correction is imperative at an early age, and treatment should be started as soon as the deformity has manifested itself. An early recognition of dental cripples by the pediatrician is to be desired. I therefore sincerely hope that you, as a group, will bring the matter before the medical school authorities in the hope that a few lectures on orthodontia diagnosis may be included in the medical school curriculum in the near future.

I am sorry to say that we are greatly handicapped in our work by lack of knowledge in determining causes of certain deformities. If we knew more about the etiology, I am sure fewer failures would result. However, we do know that these dental cripples exist and that they can be helped, provided they are not permitted to remain crippled beyond the remediable age. It is not my intention to leave with you the impression that we are not constantly endeavoring to seek this knowledge, for we have at the Harvard Dental School, a Fellowship in Research conducting experiments which will, I am sure, throw great light on many of our complex problems, for example, exactly what happens when very narrow, high arches are developed. Reflexes are being studied which will help us greatly with our reflex habit cases.

The pediatrician, the rhinologist and the or-

thodontist are vitally interested in growing children and all should strive to keep them normal so that nothing will interfere with proper function. A closer coöperation of all three is to be desired.

Discussion

Chairman O'Keefe: This paper will be discussed by Dr. Richard M. Smith, of Boston.

Dr. Smith: *Mr. Chairman, Ladies and Gentlemen*—I like particularly the term which the speaker has taken for his discussion as applied to these children with bad occlusion as "dental cripples" because we are concerned then with preventing abnormality, which is a much broader point of view than simply attempting to move teeth about in the jaw, and it raises the question to a very much higher level of preventive therapeutics. Just as soon as we realize that there is a very close association between malocclusion and the bony development of the jaw, we feel that we are dealing with an important factor in general health, and that we have removed it from the field of strictly dental treatment.

The extensive mechanical procedures which were frequently used in correcting conditions of malocclusion, such as have been discussed frequently, involve long-time treatment and sometimes, less frequently now than formerly, the use of complicated apparatus. Not infrequently this long-continued treatment has a definite effect upon the general health of the child, an effect unfavorable sometimes in its physical manifestations but more frequently in its nervous results. It is only as we can appreciate that this work has a wider usefulness than a purely esthetic one that we are willing to make the necessary adjustments in the child's life and routine in order that these corrective procedures may go forward. It is necessary frequently to make temporary sacrifices at least in other directions in order that this work may be carried on effectively.

Fortunately, with the development of better procedures on the part of the orthodontist, the duration of the treatment and the severity of the mechanical appliances used are much less than formerly; but still problems not infrequently arise in which we have to determine just how much sacrifice in other directions must be made in order to carry out this procedure. And, as I said a moment ago, when we realize that it has a real health value, we are willing then perhaps to make more sacrifice than we would be willing to make under other conditions.

I am very glad that Doctor Bonin emphasized the importance of undertaking treatment of this sort early. You notice he said that we should undertake corrective treatment as soon as the deformity was recognized. I hope that either he or Doctor Rogers will say a little more about the exact time and how we shall recognize these cases early. He has explained that somewhat and shown us one or two illustrations but I do think that it is important for us to realize that we should not wait until the crippling has gone on for any considerable period of time before we undertake corrective measures, because these cases that are postponed are the ones that require long and difficult treatment. The ones that are recognized early are the ones that can be treated for a relatively short period of time and with relatively simple apparatus and relatively little strain upon the child.

I think it is also perhaps pertinent to emphasize again the point which was made at the close of the reader's paper, namely, that all of us, physicians particularly who are in practice with children, need to be educated in this field, and this education certainly should begin in the medical schools, and it ought to continue throughout our professional life, in order that we may be apprised of the developments in this field and may be made to realize the importance from the general health point of view of prevention of dental cripples.

Chairman O'Keefe: The paper will be discussed further by Dr. Alfred P. Rogers of Boston.

Dr. Rogers: *Mr. Chairman and Members of the Pediatric Section*—It gives me great pleasure to be privileged to speak to you. In discussing Dr. Bonin's paper let me say first of all that I see in this occasion a very hopeful sign. Orthodontia, as you know, is a new profession. It is an offshoot of dentistry, but fundamentally it has less in common with dentistry than with pediatrics.

In considering the above statement we must remember that the teeth in number, size and form are determined the moment the germinal cells unite. During the development of the individual, prenatal or postnatal influences seem to have little effect upon the size and form of the teeth; but frequently as the child advances in years it is found that the skeletal development is in some manner interfered with so that it may appear arrested or malformed. This is true not only of the facial bones themselves, but also of the alveolar tissue.

It may, therefore, be seen that orthodontic problems are not merely dental but are essentially problems of growth and development; and being such must be considered very much in your field.

We, as a profession, feel in need of medical support and guidance. We find at work a great many etiologic factors which baffle us, and we are compelled to confess that we are not at present in a position to delve deeply enough into the basic causes of malocclusion. Therefore, in coming together in this fashion we

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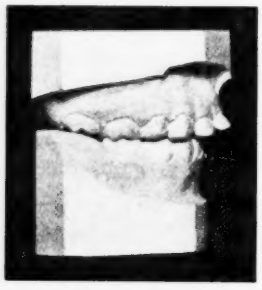

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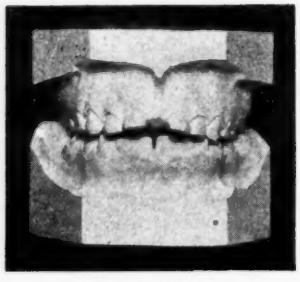

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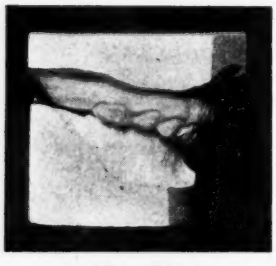

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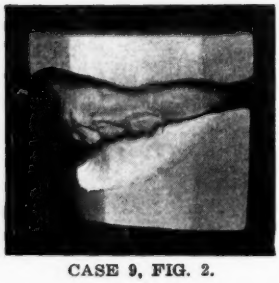

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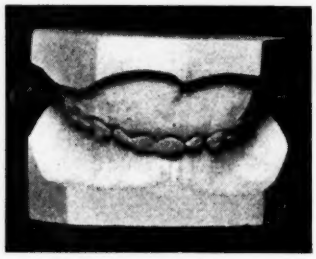

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stimulate a mutual interest in our common problem.

The suggestion regarding the question of diagnosis made by Dr. Bonin is important. The pediatrician examines the child's body in its completeness, and it is necessary that he know all parts of the body in health and in disease. In order to diagnose correctly, the pediatrician must have an understanding of the maldevelopments taking place in the facial form of the child. I would, therefore, agree with the suggestion that the pediatrician become conversant with the principles of orthodontic diagnosis. It might not be necessary that many lectures be given. The pediatrician could have at hand a proper diagnostic pamphlet designed to instruct, on very short reading, exactly how to go about the task of diagnosis. At the same time proper terminology could be mastered.

There is little criticism that I would make of Dr. Bonin's paper. I think he has discussed it in a very clear way so that it is easily understood. The terminology perhaps might have been a little different. The fact is we do not now use the terms Class I, Class II and Class III so frequently as we did a few years ago. We now find that in the development of diagnosis we are developing also a new terminology so that the Class I, Class II and Class III divisions are gradually disappearing from our literature, and a more scientific terminology is taking its place.

I should like to call your attention to a few of the problems we would wish you to solve for us. The question of nutrition is one of great importance. It is disconcerting to the orthodontist that children who seem to be properly nourished still show signs of developing malocclusion. I have in my practice identical twins whose divergent oral development has completely upset my theory regarding nutrition. During the early years one of these children suffered from a gastrointestinal disturbance which made it necessary for her to live on a rather restricted diet, spending much of her time in bed. Her sister was more robust and enjoyed the ordinary privileges of childhood as to play, diet, etc. To my amazement the ailing child, the one confined to bed upon a restricted diet, is blest with splendid facial development, while the one who received the well-rounded diet and appeared much the more normal of the two has developed rather a bad form of malocclusion.

One pertinent point which Dr. Smith made in his discussion relates to early treatment. For many years I have felt the wisdom of this course, but these years of experience have taught me caution. As you well know development in children takes place at different periods, that is, the physiologic age varies, particularly regarding oral development. I have been gratified at times in studying children under observation to find the necessity for treatment postponed from time to time where a gradual improvement manifested itself.

I have observed during my life's experience that children tend to grow and develop much more normally between the months of March and October. It is, therefore, my practice to give children comparative freedom during that part of this period which includes their summer vacation, allowing them to go unhampered so far as possible. Inherent growth tendencies then have an opportunity to assert themselves. I have seldom been disappointed. It is my opinion that this period should be thoroughly studied by pediatricians, orthopedists and orthodontists. My thought then is that the idea of early treatment is fundamentally sound, but that it may be, and has been, overdone much to the detriment of many children.

There is another problem that confronts us, and I am sure you will appreciate it and sympathize with us. Our profession, as I said a short time ago, is young. During this period of its development unfortunately any person who has obtained a dental grade and has passed his State examinations is entitled to practice orthodontia, yet he may not have the slightest ability, nor may he have sufficient knowledge of skillful practice. A very large number of cases coming under the notice of pediatricians are not being treated by well-trained orthodontists. This is a condition that we will find difficult to overcome. Some day we may need your support. It should not be possible at this advanced stage of progress, for anyone, except those properly qualified, to undertake difficult orthodontic procedures which sometimes have far-reaching effects. You can understand, therefore, that this problem needs our attention.

CHAIRMAN O'KEEFE: This paper will be further discussed by Dr. Lawrence W. Baker of Boston, who has some exhibits and slides.

DR. BAKER: *Mr. Chairman, Ladies and Gentlemen*—It is needless to say that, being a co-worker with Dr. Bonin, I am much gratified with the interest you have shown in his paper, and am particularly pleased with Dr. Rogers' discussion as a co-worker as well as with Dr. Smith's broad discussion of it from a medical standpoint.

Dr. Bonin had a good deal to say about the effects of widening the arches on the bones of the head. For a long time we orthodontists have not known what takes place when the arch is widened. One group thought the change was only in the supporting bone of the teeth, the alveolar process. It was the opinion of another group that aside from the alveolar change there was also a bony change in the palate, particularly along the suture between the maxillary bones. Still another group thought that, because of the great general improvement in those children whose collapsed dental arches had been expanded, the bony development must be far-reaching, involving not only the maxillary

bones but many other bony structures of the face and head. One man's opinion, however, is as good as another's, because all this was mere hypothetical reasoning.

At present I am at work on an investigation which I hope will show just what bony changes do take place in arch expansion. Day before yesterday I completed a preliminary experiment in this course of investigation which seems to throw sufficient light on the problem for me to venture to present it to you this morning.

In these investigations I have gone back one hundred and fifty years to John Hunter. I am employing the same principles of vital staining with madder used by this great genius. It was by the use of madder staining that Hunter forced Nature to reveal some of her deepest secrets about growing bone. Hunter knew that when madder was fed to animals it stained growing bone, and only bone that was growing. He knew that in some mysterious way madder was deposited in bone during the process of calcification. He therefore fed growing animals madder for a definite period of time and then stopped the madder feeding and allowed the animals to grow to maturity. Only the bone that had grown during this period was, of course, stained.

Undoubtedly you have all seen in the Royal College of Surgeons in London those classic experiments just referred to in which Hunter caused nature to print for him the bones of young pigs upon the same bones of the adult animal. You will recall that the bone which was deposited during the period of madder feeding was stained red while the bone which developed prior to and after the madder feeding was white.

In my investigations I am applying Hunter's principles, but in a somewhat different manner from the way he used them. I am studying bone growth stimulated by applied force. Accordingly, I am working not with growing animals but with adult animals after the skeleton has completely developed. I am, therefore, putting appliances on the upper arches of adult cats and expanding the arches precisely as Dr. Bonin expanded the arch of the child.

Much to my surprise, I found, as this specimen shows, that there was not only a change in the alveolar process and palate particularly along the suture, but that the internal and external bones of the face as well were also distinctly reddened. There was also an indication that the change had even extended along the interparietal suture. The greatest change, however, took place nearest the application of force, which was in the maxillary region. The rhinologists present will be particularly interested to note that the bones of the nasal cavity and floors of the orbits are deeply stained, especially along the sutures.

The specimen was then passed around. Dr. Baker emphasized the fact that this was but a report of progress and pointed out that although this preliminary experiment fulfilled Hunter's law, nevertheless to prove these facts conclusively the investigation is being carried farther. He further stated that his researches are being carried on at the Harvard Medical School in the departments of pharmacology and anatomy and that the heads of both departments, Dr. Reid Hunt and Dr. George B. Wislocki, have given him invaluable aid.

Dr. Baker then amplified Dr. Bonin's remarks with a description of a series of slides illustrating the changes in the facial lines brought about by orthodontic treatment. A few of these cases treated in the clinic of the Harvard Dental School are here reproduced.

Figure 1A shows the condition at the time of beginning treatment. It is a typical illustration of the distal misplacement of the lower jaw, complicated by a narrowed upper arch. The primary cause of this deformity was probably due to the blocking of the breathing spaces by enlarged adenoids and diseased tonsils. These obstructions were removed before beginning orthodontic treatment.

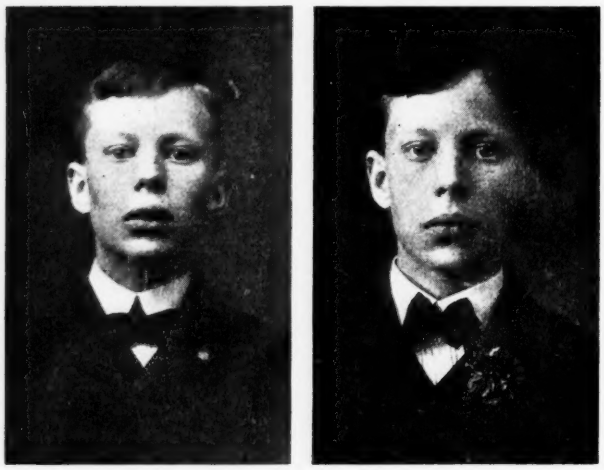
Figure 1B shows the transformation due to orthodontic treatment. As Dr. Bonin has made clear to you, these benefits have been brought about by the widening of the upper arch, the reduction of the protruding superior incisors coincidental with the bringing of the receding lower jaw forward to its normal position. The boy now has full power of his masticatory apparatus and has normal tongue room. The remodeling of the dental apparatus makes it possible for the boy to keep his mouth closed without conscious effort.

Figure 2 illustrates the reverse of the conditions in Case 1. A shows the influence of the protruding lower jaw on the facial lines. B shows the result of treatment. This case was cared for by Drs. Fred R. Blumenthal and Shinji Fujishiro.

The next case, shown in figure 3, is an illustration of the same type of case as shown in figure 1, viewed from the side.

This case is presented as an example of the result of coöperation of the rhinologist and the orthodontist. Although the rhinologist cleared the nasopharynx of the obstructions, the dental deformity shown in figure 3A made it impossible for the boy to close his mouth except by conscious effort. He would, therefore, undoubtedly continue to be a chronic mouth breather. The orthodontist, on the other hand, could not bring about a complete cure without the aid of the rhinologist. The result of the combined treatment of both is shown in figure 3B.

Let us hope that Dr. Bonin's paper will have a far-reaching influence in bringing about a

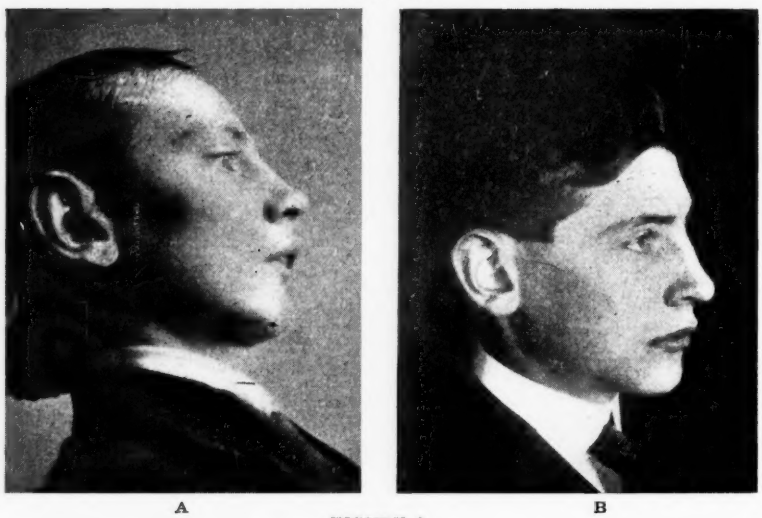


A B

FIGURE 1.

A B

FIGURE 2.



A B

FIGURE 3.
ILLUSTRATIONS OF PATIENTS REFERRED TO BY DR. BAKER.

closer coöperation between medicine and dentistry.

DR. BONIN: *Mr. Chairman*—The only point I want to speak of is in regard to the inquiry of Dr. Smith as to how early we should undertake to treat these young cases. I fear that I gave the wrong impression when I said we should start all young cases just as soon as a marked deformity was apparent. What I meant by that was a deformity where the mandible has been misplaced either backward or forward. I do not believe that any such condition will rectify itself, because if not treated it will become a reflex condition. After the mandible is displaced forward or backward a habit has been formed and that child does not know that his jaw is not closing properly, so consequently it must be given a new way of functioning, which might be

properly termed a functional reflex. There is no better way to do that than to bring that jaw forward or put it back where it belongs so that proper interlocking of the teeth will take place. When that has been established you have normal function and a beautiful development follows, whereas if the deformity were not corrected the muscles would be pulling the wrong way, which would aggravate conditions. I would also advise early treatment of a marked protrusion of the maxilla which almost invariably develops into a tongue habit condition.

I showed a case of a child three years old which I believe is the youngest case I ever started. As I pointed out, the lower jaw was misplaced forward, and in three months his jaws were made normal. All appliances were then removed and from that point on nature was given a chance to do her work. If the child had been allowed to go until he was ten or twelve years old he might have been treated successfully, but then again he might not; I have my doubts. It is much easier to correct conditions at an early age and thus secure normal function so that when the appliances are removed the jaws will continue to develop as they should.

I cannot close without expressing my appreciation of the way in which my little contribution has been received. I wish particularly to thank Drs. Smith, Rogers, and Baker who discussed it in such a broad manner.

CHAIRMAN O'KEEFE: We will listen now to the report of the Nominating Committee.

DR. RICHARD M. SMITH: *Mr. Chairman*—The Nominating Committee presents for officers of this Section for next year the following:

Chairman: Dr. Gardner N. Cobb, Worcester.
Secretary: Dr. James M. Baty, Boston.

CHAIRMAN O'KEEFE: You have heard the nominations. What is your pleasure?

Upon motion duly made and seconded, it was

Voted: To close nominations, and that the Secretary be instructed to cast one ballot for said nominees.

The Secretary having cast the ballot as directed, the Chairman declared Dr. Cobb and Dr. Baty elected to their respective offices for the ensuing year.

CHAIRMAN O'KEEFE. The next paper is on the subject of "Vitamin D Milk," to be read by Dr. Edwin T. Wyman, Boston, Instructor in Pediatrics at Harvard Medical School.

VITAMIN D MILK*

BY EDWIN T. WYMAN, M.D.†

THE development of vitamin D milk has been the result of careful scientific coöperation between workers in research laboratories, clinicians, and dairymen.

Hess[1] in 1927, studying various foods, reported that yeast acquires a high antirachitic potency when irradiated with ultraviolet light. Following this, the possibility of producing milk rich in vitamin D by feeding irradiated yeast to cows was reported in Germany by Wachtel[2] and in this country by Steenbock and his coworkers[3] of the University of Wisconsin.

In 1930 Thomas and MacLeod of the Walker-Gordon Laboratory Company fed irradiated yeast and irradiated ergosterol in varying amounts to different groups of cows, and tested the vitamin D potency of the milk produced. They reported[4] that to get milk of a given vitamin D potency it was necessary to feed twice as many units of vitamin D in the form of irradiated ergosterol as in the form of irradiated yeast. The Fleischmann Laboratories coöperated in this work by supplying irradiated yeast of standardized vitamin D potency and in determining the vitamin potency of the milk produced. Laboratory experiments showed this milk to contain approximately 160 vitamin D units per quart.

Laboratory estimations of the potency of vitamin D milk were made by feeding to rachitic rats weighed amounts of the pure fat from a sample of the milk under examination and determining the amount of fat required to produce a given degree of healing in the so-called "line test" as outlined by the Wisconsin Alumni Research Foundation.

The potency of vitamin D milk is expressed in terms of the Steenbock unit which is defined as the total amount of vitamin D, which, fed over a ten-day period will produce a narrow continuous line of healing, termed a 2+ line.

During the past year the production of vitamin milk (160 units per quart) has extended to certified farms in Boston, New York and Philadelphia. It is a measure that is especially applicable to the certified farms where the ration of the cows is thoroughly controlled and one can be certain that a potent yeast is fed in proper amounts, and more particularly where frequent biologic assays of the milk can be carried out.

The purpose of this paper is to review the clinical evidence thus far available for apprais-

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Read before the Section of Pediatrics of the Massachusetts Medical Society, June 7, 1933.

†Wyman—Instructor in Pediatrics, Harvard Medical School. For record and address of author see "This Week's Issue," page 920.

ing the antirachitic potency of such vitamin D milk.

Hess and his associates[5] in 1931, fed the vitamin D milk to more than 100 babies, some of them normal and some suffering from rickets. In all cases in which milk having approximately 160 units per quart was used, a daily average of a pint and a half per infant not only was found ample for complete protection against the development of rickets but also healed rickets within a month, in the cases in which rickets was present.

Yeast Milk which contained approximately 100 units per quart was supplied to Drs. H. J. Gerstenberger and A. J. Horesch[6] of the Babies' and Children's Hospital in Cleveland. When fed to rachitic babies in amounts of one pint per day, steady improvement was obtained. This improvement was evidenced both by Roentgenographic examination and blood studies. The experiments, although conducted with only two children, gave definite evidence that 50 units of vitamin D in this form effectively healed rachitic lesions, even though the infants received only approximately 50 units of vitamin D daily.

In a study at the Infants' Hospital in Boston we[7] also obtained proof of the curative value of vitamin D milk. The complete evidence for one case obtained with the coöperation of Dr. Butler follows.

D. E. aged 16½ months, one of the patients studied in this group, was admitted to the Infants' Hospital with active rickets. The patient was kept in bed on the ward throughout the entire time of ob-

TABLE 1

SUMMARY OF CASE 1, D. E., AGED SIXTEEN MONTHS

Serum, calcium and inorganic phosphorus values and x-ray findings during the course of the experimental period.

Date, 1931	Weight, Pounds	Calories per Pound in Diet	Serum Calcium Mg. per 100 Cc.	Serum Phosphorus Mg. per 100 Cc.	Calcium Phosphorus Product	Roentgenograms
March 31	18.0	45.0	10.4	3.3	34.3	Advanced active rickets: slight deposition of tricalcium phosphate in osteoid tissue
April 13	18.0	45.0	10.7	2.5	26.8	No further change
April 23*	18.3	43.8	9.7	2.4	23.3	No further change
May 5	18.7	51.6	11.8	5.5	64.9	Healing rickets
May 18	19.5	49.5	11.4	5.9	67.3	Further healing
June 9	19.6	49.1	11.5	5.7	65.6	Further healing

*Special milk started.

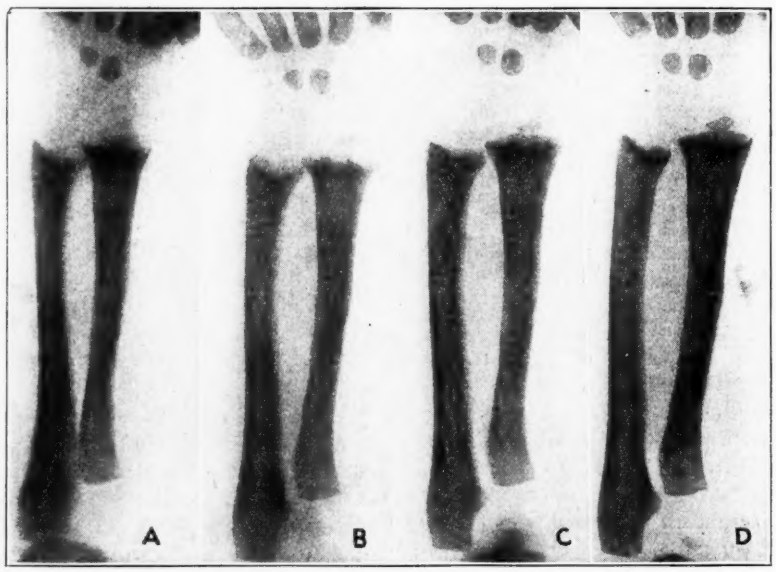

FIG. 1 (Case 1). Roentgenograms of the radius and ulna of D. E., aged 16 months. *A*, taken March 31, showing advanced active rickets with slight deposition of tricalcium phosphate in osteoid tissue; *B*, taken April 23, showing no further change during control period; *C*, taken May 9, after sixteen days on the special milk, showing healing rickets, and *D*, taken June 9, showing further healing.

servation. Roentgenograms were taken and serum calcium and inorganic phosphorus determinations made in order to follow the progress of the therapy. The patient was placed on a metabolism bed during the control and vitamin D milk periods and studies were made of the phosphorus and calcium balances. The first period was during the time of observation without vitamin D and the second and third periods while vitamin D milk was being given.

The calcium phosphorus product at entry was 34.3. Roentgenograms showed a slight deposition of opaque salts in the osteoid tissues at the end of the diaphyses. During the control period the calcium-phosphorus product dropped (table 1) so that on April 23 it was 23.3. In the three week control period the inorganic serum phosphorus fell from 3.3 mg. to 2.4 mg. per hundred cubic centimeters. This is evidence against the presence of any significant amount of latent rachitic healing following interrupted therapy. For, as shown by Gerstenberger, Hartman, Russell and Wilder[8], had such healing been present, the serum phosphorus should have risen. The roentgenograms taken during the control period also showed no detectable change. Therefore, we felt that latent healing following interrupted anti-rachitic therapy could be ruled out, and we considered this case suitable for study. On April 23, 1931, pasteurized vitamin D (yeast 160 units) milk was substituted for the ordinary milk of the diet. On May 5, the amount of milk was increased from 32 to 40 ounces (946 to 1183 cc.). Except for these changes, the routine diet was maintained constant during the ten weeks of hospitalization.

In table 1 are presented the serum calcium and inorganic phosphorus concentrations and the roentgenographic findings.

Figure 1 gives the reproduction of four of the roentgenograms from this patient.

The marked change in the inorganic serum phosphorus, the elevation in the calcium and phosphorus product, and the roentgenographic evidence of healing rickets indicate the effectiveness of this form of vitamin D therapy.

Table 2 prepared by Butler[9] presents the calcium and phosphorus balances on this patient during the period of study. The table includes data on patients with active rickets, one treated with cod liver oil and the other with ultraviolet radiation as reported by Schabad[10] and Orr[11].

It will be noted from table 2 that the positive calcium balance of 0.078 Gm. during the control period increased to 0.855 Gm. during the last vitamin D milk period, while the positive phosphorus balance increased from 0.020 Gm. to 0.592 Gm. during the same period. In the control period 6.7 per cent of the calcium and 2.2 per cent of the phosphorus ingested was retained as compared with a retention of 56.4 and 52.2 per cent respectively in period III. These increases in positive balances compare favorably with the results obtained by the other investigators using cod liver oil and ultraviolet light as therapeutic agents. It is an interesting fact that excretion of phosphorus in the urine remained nearly constant in all three periods of the experiment in spite of the marked change in the inorganic phosphate concentration in the

TABLE 2

CASE 1, D. E.

Author and Period	Gm. Calcium per Day				Gm. Phosphorus per Day				Weight in Kilos
	Food Intake	Urine	Stool	Total Excretion	Balance	Food Intake	Urine	Stool	Total Excretion	Balance	
Butler											
Period I	1.160	0.002	1.080	1.082	+0.078	0.912	0.290	0.602	0.892	+0.020	8.2
Period II	1.216	0.000	0.950	0.950	+0.266	0.914	0.406	0.371	0.777	+0.137	8.2
Period III	1.516	0.008	0.653	0.661	+0.855	1.132	0.382	0.158	0.540	+0.592	8.8
Schabad											
Rickets untreated	1.140	0.001	0.910	0.911	+0.229	1.060	0.330	0.590	0.920	+0.140	
After C. L. O.	0.980	0.003	0.260	0.263	+0.717	0.820	0.340	0.120	0.460	+0.360	
Orr et al.											
Rickets untreated	1.438	0.011	1.452	1.463	—0.025	1.059	0.296	0.764	1.060	—0.001	9.6
After ultraviolet	1.289	0.042	0.544	0.586	+0.703	0.946	0.315	0.185	0.500	+0.446	9.6

Average daily balance data of Case 1 for each of the three six-day periods together with similar data from two cases of infantile rickets, one treated with cod liver oil and the other with ultraviolet light as reported in the literature.

serum. The greatest change in the excretion of both calcium and phosphorus during the three periods of investigation was found in the stool.

CONCLUSIONS

1. Vitamin D (yeast 160 units) milk has certain advantages in infant feeding. The administration of antirachitic vitamin in so palatable a form and the automatic prevention of the occurrence of rickets relieve the physician of anxiety as to how much and what antirachitic agent to give.

2. Vitamin D (yeast 160 units) milk while it prevents and cures rickets can do no harm. We have continued many babies on this milk for the second year and found that they thrive excellently well and that none of them developed rickets in the course of two winters. Evidence is accumulating that an antirachitic supplement is required throughout childhood during growth and development.

3. Vitamin D (yeast 160 units) milk should be given during the summer months as well as the winter months as the amount of sunlight the average young growing baby gets during the summer months in this latitude cannot be relied upon to prevent the occurrence of rickets.

4. Further investigations should be made before the potency of the vitamin content of milk advertised as rickets curing is lowered, although 50 to 100 units per day in milk may be all that is required, for in young rapidly growing babies most needing vitamin D the milk intake is only one and one half to two ounces per pound of body weight. The 160 unit amply supplies this need, but a milk of lesser potency might not be adequate.

5. Further investigations should be made to draw a reliable comparison between the efficacy of activated milk and that of yeast milk. It seems reasonable to believe that yeast milk will continue to be produced by certified farms and that irradiation of milk can be made a part of the pasteurizing process on lower grades of milk.

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DISCUSSION

CHAIRMAN O'KEEFE: This paper will be discussed by Dr. Lewis W. Hill, of Jamaica Plain.

DR. HILL: *Mr. Chairman*—Dr. Wyman's work has shown beyond question that vitamin D certified milk will cure rickets. Doctor Hess' work has shown that it will prevent rickets. We have therefore something which really works. Is this milk simply a rather interesting curiosity, or is it something that we should all take unto ourselves and use for general purposes of infant feeding? What are some of its advantages and some of its possible disadvantages?

In the first place, it is the cleanest and safest milk that can be produced. The bacteria count of certified milk, raw, will run from 1,000 to 3,000. The bacteria count of certified milk pasteurized will run from 100 to about 300. That is practically bacteria-free. Certified vitamin D milk is produced under the best conditions in every way, and no effort is spared to make it the finest possible milk.

Secondly, it will prevent rickets without the addition of any other antirachitic agent. The mother is therefore relieved of one extra procedure in the care of her baby. She needs to give no viosterol or cod liver oil. It protects against rickets automatically without any thought on the part of the mother. It protects against rickets by supplying the antirachitic factor in the natural food supply instead of giving artificial additions.

Thirdly, the baby smells like a baby and not like a codfish. I think it is fair to say that, leaving the question of cost out of consideration, vitamin D milk is the very best possible milk that we can use for the general purposes of infant feeding. It costs, however, twenty-five cents a quart. Grade A milk costs fourteen cents a quart. A month's supply of vitamin D certified milk costs $7.50. A month's supply of Grade A milk plus viosterol, if the viosterol is bought in the five cc. bottles, costs $6.45 a month. A month's supply of Grade A, plus viosterol if bought in the 50 cc. bottles, costs $5.30 a month. A month's supply of Grade A, plus cod liver oil, costs $5.10 a month. There is, therefore, a difference of $2.40 a month between vitamin D and Grade A plus cod liver oil. If the family budget will stand it, it seems to me that vitamin D milk is the best milk to use for general infant feeding purposes. It has a good many advantages. If the family budget will not stand it, the mother will do much better to stick to Grade A plus cod liver oil.

DR. RICHARD S. EUSTIS, Boston: You may be interested in the supervision that the Medical Milk Commission of Boston maintains over the production of vitamin milk on its four certified farms.

The interest of the Commission has been in making certain that the product came up to the advertised standard of vitamin D content. We, therefore, leave to the farm the details of feeding irradiated yeast of known vitamin D content to the cows in amounts proportionate to their yield of milk. We collect samples of vitamin milk at irregular intervals without previous notice to the producers and deliver them to the Massachusetts Institute of Technology for bio-assay. These assays are made according to standard methods on groups of young rats on a rickets-producing diet. Four animals are kept as a control and eight are fed measured amounts of whole vitamin milk over a ten-day period. Then all the rats are killed and the degree of healing of the rickets is determined by examination of the epiphyses of the femurs. All samples tested to date have contained over 160 Steenbock units per quart.

Irradiated yeast is now being sold to some producers of Grade A milk. No public health authority has the laboratory facilities to test the vitamin D content of its product. The distributors of the yeast are trying to work out a method of control by comparing the amount of yeast purchased by the farm with the number of milch cows and the amount of milk produced. This does not seem to me a sufficiently accurate and certain method for doctors to rely upon, without the confirmation of occasional bio-assay.

In the practice of medicine, we have learned the need of being sure of the potency of the medicines that we use, and certainly vitamin D milk must be regarded as a medicine. We believe that through our supervision the vitamin D content of vitamin D certified milk is as definite and as much to be relied on as is the vitamin D content of cod liver oil, viosterol or halibut liver oil.

However, the vitamin D content of a food or a drug as measured in Steenbock units is not the only point to be considered. Hess has pointed out very clearly in several of his papers the great variation in rat units of a number of preparations of approximately equal clinical value. He states that a satisfactory preventive dose of vitamin D for an infant is ten drops of viosterol, 800 units; or three teaspoonfuls of cod liver oil, 200 units; or 24 ounces of yeast milk, 120 units; or 24 ounces of irradiated milk, 35 units. In these amounts, viosterol, cod liver oil, yeast milk and irradiated milk appear to be of equal value to the human being but not to the rat. In spite of this discrepancy, the rat unit is the only practical method we have at present for measuring the vitamin D potency of a substance. It would seem that bio-assays on rats are of value in maintaining a standard of potency in each vitamin D containing food or drug, but that clinical experience must determine what that standard should be for each food or drug.

As a practical matter, what we are interested in is the convenience and palatability of the form of vitamin D that we select for the individual patient. Vitamin D milk has been shown to be a satisfactory method of preventing and treating rickets, is very little more expensive than Grade A milk plus cod liver oil or viosterol, is palatable, and is fairly certain to be taken by the patient.

CHAIRMAN O'KEEFE: If there is no more discussion, Dr. Wyman will close.

DR. WYMAN: I have nothing further to add.

CHAIRMAN O'KEEFE: Then we will listen to "The Preservation of Human Milk," by Dr. Paul W. Emerson.

THE PRESERVATION OF HUMAN MILK*

VII. The Feeding to Premature Babies of Human Milk Preserved by Freezing

BY PAUL W. EMERSON, M.D.†

A PROCESS of preserving human milk has been described in a preliminary note[6] which compares favorably with other methods which have been used[1, 2, 3, 4, 5]. This preparation of "frozen" milk, by which we mean, throughout this paper, milk that has been preserved by freezing and liquefied before feeding, does not differ from that of fresh milk in total solids, fat, carbohydrates or protein. Bacterial examination disclosed bacterial counts which were surprisingly low. It has been shown that the vitamin content of fresh human milk is not changed by this process of freezing. Nine premature babies who were fed on the milk gained in as satisfactory a manner as they would on fresh milk. From these preliminary observations it was concluded that human milk preserved by freezing could be used effectively as a food for infants.

Recently, sixteen premature babies have been

*From the Research Laboratory of the Directory for Mothers' Milk, Inc. and the Department of Pediatrics, Harvard Medical School.

Read at the meeting of the Pediatric Section of the Massachusetts Medical Society, June 7, 1933.

†Emerson—Instructor in Pediatrics, Harvard Medical School. For record and address of author see "This Week's Issue," page 920.

observed, making now a total series of twenty-five infants who have been fed "frozen" milk. Twelve patients were observed at the Boston Lying-In Hospital†, with three babies fed on fresh milk serving as a control series. The remaining thirteen babies in this series were observed at the Infants' Hospital. The oldest baby was one-half month premature, the youngest three months premature. Eight were males and seventeen females. The weight at birth averaged 3.1 pounds; the largest baby weighed four pounds and thirteen ounces and the smallest baby weighed two pounds. The patients varied in length from fourteen to twenty inches, the average length being seventeen inches. In most cases the body temperature became stabilized at three weeks of age. During the period of observations nine babies showed clinical signs of atelectasis, three had otitis media, two had thrush, three a dermatitis, one an infected finger, one an umbilical abscess, one a conjunctivitis and one a birth injury and hydrocephalus. One baby was gravely ill with frequent attacks of cyanosis for the first few days. One other baby who had advanced syphilis was critically ill on entry.

Babies on Fresh Human Milk	Babies on Frozen Human Milk	Time on Fresh Human Milk in days	Gain in oz.	Average Daily Gain in oz.	Time on Frozen Human Milk in days	Gain in oz.	Average Daily Gain in oz.	Fresh H. Milk: Feeding in Days by Gavage or Breck	Refusal, No. of Times	Vomiting, No. of Times	Stools	Average	Caloric Intake	Frozen H. Milk: Caloric Intake	Feeding in Days by Gavage or Breck	Refusal, No. of Times	Vomiting, No. of Times	Stools	Average
	Ca				20	9	.5							63	1	0	2	4-0	2+
	Mu				62	33	.5							60	28	0	6	4-0	1-
	Cov				35	23	.7							65	14	0	12	4-1	2-
	Bu				84	33	.4							66	35	0	14	7-0	2
	Fr				99	44	.4							66	28	0	28	7-0	2
	Ct				46	23	.5							61	0	0	0	5-0	2
	Mac				11	12	1.0							74	6	10	4	5-3	3
	Ci				24	16	.7							60	0	2	0	6-2	4
	Ja				15	7	.5							61	0	7	4	5-2	4
	Bus				42	26	.6							67	12	35	11	5-3	3+
	Sh				34	20	.6							65	14	0	8	6-1	3
	T#1				31	17	.8							62	15	10	3	6-3	3
	La				20	10	.5							64	0	8	2	6-2	4
	Ma				41	25	.6							66	0	11	4	5-3	3
	H#1				18	12	.7							61	0	3	0	5-1	4
	Ke				21	10	.5							65	0	7	0	5-1	3
Total					593	320									153	93	104		
Average							.54								17	10	8		
WKn	WKn	24	5	.2	37	23	.6	0	0	6	7-1	3	61	61	0	0	0	6-3	3
RHa	RHa	36	21	.6	27	13	.7	0	0	0	6-1	3	60	61	0	0	0	6-0	2
Bl	Bl	14	4	.3	43	14	.3	0	0	0	5-0	2-	65	62	0	0	7	8-5	4-
Br	Br	14	6	.4	28	11	.4	5	0	4	8-1	5	69	64	15	0	1	6-0	1+
CWr	CWr	27	15		29	15	0	14	0	0	5-2	4	0	0	0	0	7	4-1	3
CLv	CLv	45	49	1.0	12	4	.3	0	5	1	6-0	3	70	66	0	11	1	6-2	3+
LeB	LeB	15	18	1.2	15	8	.5	4	5	2	6-2	4	57	63	14	20	1	5-2	4
MPa	MPa	14	9	.6	26	18	.7	3	7	1	5-2	3+	63	65	0	2	0	6-2	4
Total		189	127		217	111		26	17	20					29	33	23		
Average				.67			.51	7	6	3					12	11	4		
Grand Total					810	431									182	126	127		
Average							.53								17	10	7		
OD		32	24	.7				35	0	25	6-0	3-	66						
T#2		36	24	.7				21	9	11	5-1	4	66						
H#2		18	10	.6				10	14	1	5-1	4	63						
NRe		34	24	.7				31	9	13	5-0	2+	67						
Total		120	82					101	32	50									
Average				.68				25	10	13									
Grand Total		309	209					127	49	70									
Average				.67				16	8	7									

TABLE II.

Baby CWr: The average daily gain in ounces on fresh human milk should read .6 instead of 0. The average daily gain in ounces on frozen human milk should read .5.

†The feeding of "frozen" milk to infants at the Boston Lying-In Hospital was under the direction of Dr. Stewart H. Clifford.

In this study the milk which had been "frozen" by the Directory for Mothers' Milk was

taken out of storage and thawed in the container, by immersing it in cold water for half an hour. The fluid milk was then packed in ice and transported to the nurseries where it was kept in electric refrigerators at a temperature of 40°F. until needed. The age of the frozen milk varied from one to 220 days and it

age at which orange juice and cod liver oil (or ergosterol) were added to the babies' milk was 4.1 weeks, the youngest baby being 2.5 weeks old when they were added, and the oldest nine weeks. Antirachitic and antiscorbutic vitamins were added at about the same time whether the infants were being fed fresh or frozen milk.

	Average No. of days Gavaged	No. of cases Gavaged	No. of cases Not Gavaged	No. of times refusal occurred	No. of cases Refusing	No. of cases not refusing	Average No. of times vomiting occurred	No. of cases vomiting	No. of cases not vomiting	No. of stools, average	Calories per lb.
Group I	16	8	4	8	6	6	7	10	2	2-5	56-70
Group II	17	11	13	16	12	12	7	19	5	1-4	60-74
Group III											
While on Fresh Milk	7	4	4	6	3	5	3	6	2	2-5	57-70
While on Frozen Milk	12	2	6	11	3	5	4	6	2	1-4	61-65

TABLE III.

Group I—All cases who were fed Fresh Human Milk.
Group II—All cases who were fed Frozen Human Milk.
Group III—Cases common to I and II, who received at one time Fresh Human Milk, at another Frozen Human Milk.

								On Fresh Human Milk						On Frozen Human Milk					
Babies Fed on Fresh Human Milk	Babies Fed on Frozen Human Milk	Time Fed on Fresh Milk in Days	Gain in Weight in Oz.	Average Daily Gain in Weight in Oz.	Time Fed on Frozen Milk in Days	Gain in Weight in Oz.	Average Daily Gain in Weight in Oz.	Days Fed by Gavage or Breck Feeder	Refusal, No. of Times	Vomiting, No. of Times	No. of Stools	Average No. of Stools	Caloric Intake	Caloric Intake	Days Fed by Gavage or Breck Feeder	Refusal, No. of Times	Vomiting, No. of Times	No. of Stools	Average No. of Stools
	T. No. 1				21	17	.8							62	15	10	3	6—0	3
T. No. 2		36	24	.7				21	9	11	5—1	4	68						
TABLE IV																			
	H. No. 1				18	12	.7							61	0	3	0	5—1	4
H. No. 2		18	10	.6				10	14	1	5—1	4	63						
TABLE V																			
	Cov.				35	23	.7							65	14	0	12	4—1	2—
C. Lv.		45	49	1				0	5	1	6—0	3	66						
TABLE VI																			
	Mu.				62	33	.5							60	28	0	6	4—0	1—
N. Re.		34	24	.7				31	9	13	5—0	2+	67						
TABLE VII																			
LeB.	LeB.	15	18	1.2	15	8	.5	4	5	2	6—2	4	57	63	14	20	1	5—2	4
M. Pa.	M. Pa.	14	9	.6	26	18	.7	3	7	1	5—2	3+	63	65	0	2	0	6—2	4
TABLE VIII																			

was fed to the infants over periods ranging from four to ninety-nine days, the average being thirty-four days. The bacterial counts were very low, twenty colonies per cc., for example, in milk frozen February 23, 1932 and examined August 3, 1932, five months later (table I).*

In all cases the fresh milk was pooled and pasteurized except when the baby was fed his own mother's milk; and in all cases the frozen milk was pooled and pasteurized. The average

During the fore part of this study frozen milk was fed to only the larger babies; then as it was found that they thrived satisfactorily, smaller babies were given this food. The minimum weight in one case was one pound and thirteen ounces, and in another one pound and fourteen ounces. (Charts XIV and XV.) Feeding of frozen milk was changed abruptly to that of fresh milk and vice versa, and in one case alternate feedings of frozen and fresh milk were given. Sixteen babies were fed frozen milk entirely, while nine babies were given frozen milk

*Table I appeared in the preliminary note[6] and is omitted here to conserve space.

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident No. Unit Hist. No.

Name T. No. 2. Twin of T. No. 1. Adm. No. 5766

Month ♂ Sex Birth Weight 3 lbs. 10 ozs. Length 17 inches

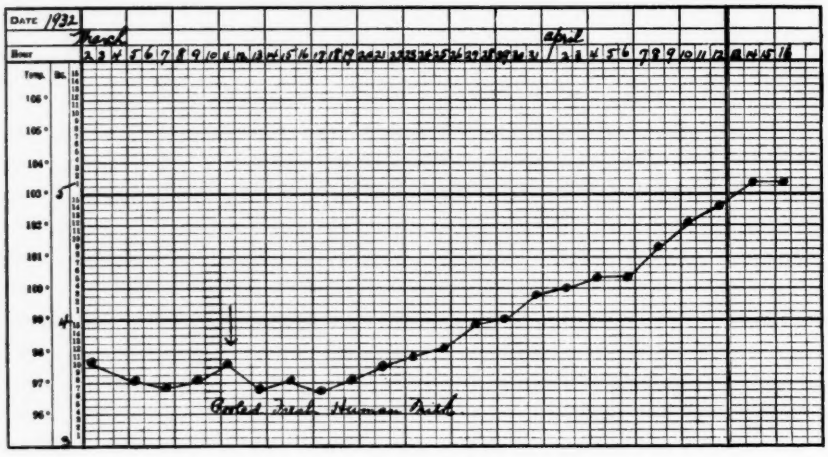

CHART I.

Premature 39 days.

Birth Weight 3 lbs. 10 oz. Length 17 inches.

Time on Pooled Fresh Human Milk 3/11/32—4/16/32 = 36 days.

Increase in Weight 3 lbs. 9 oz.—5 lbs. 1 oz. = 24 oz. Average daily gain .7 oz.

Vitamins added, for first three weeks—None.

Refusal—9 times. Vomiting 11 times, average 3 drams. Stools 5—1. Average 4.

Temperature—Stable after 2nd week.

Caloric intake 68 per lb.

Remarks. Fed by Gavage or Breck for 21 days.

Diagnosis: Prematurity.

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name T. No. 1. Twin of T. No. 2. Adm. No. 5766

Month ♂ Sex Birth Weight 4 lbs. 8 ozs. Length 18 inches

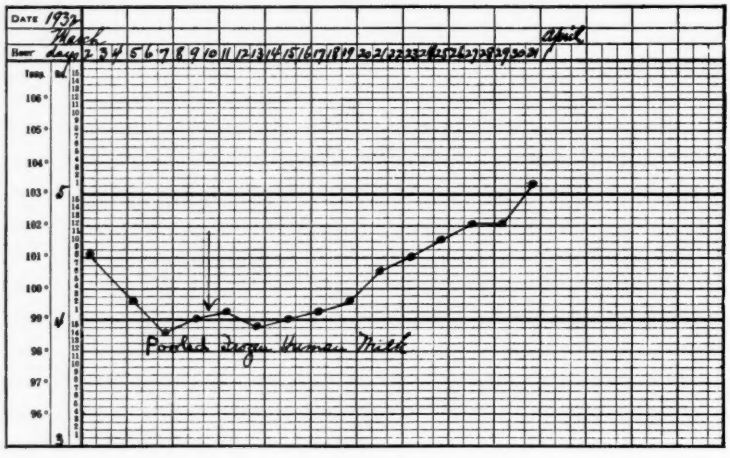

CHART II.

Premature 39 days.

Birth Weight 4 lbs. 8 oz. Length 18 inches.

Time on Frozen Human Milk 3/10/32—3/31/32 = 21 days.

Increase in Weight 4 lbs.—5 lbs. 1 oz. = 17 oz. Average daily gain .8 oz.

Vitamins added, for first three weeks—None.

Refusal—10 times. Gavage whole or in part 15 days. Vomiting 3 times. Stools 6—0. Average 3.

Temperature—Stable after 2nd week.

Caloric intake 62 per lb.

Diagnosis: Prematurity. Atelectasis (sl.).

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name H. No. 1. Twin of H. No. 2. Adm. No. 9273

Month ♂ Sex Birth Weight 4 lbs. 8 ozs. Length 20 inches

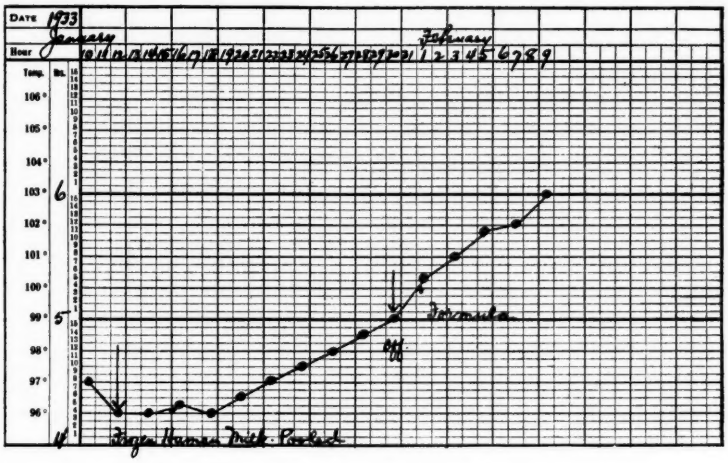

CHART III.

Premature 48 days.

Birth Weight 4 lbs. 8 oz. Length 20 inches.

Time on Frozen Human Milk 1/12/33—1/30/33 = 18 days.

Increase in Weight 4 lbs. 4 oz.—5 lbs. = 12 oz. Average daily gain .7 oz.

Age of Frozen Human Milk 92-109 days. Average 84 days.

Vitamins added, for 22 days—None.

Refusal—3 times, average 3 drams. Vomiting none. Stools 5—1. Average 4.

Temperature—Stable after 1st week.

Caloric intake 61 per lb.

Remarks. Gavage feedings first 1½ weeks.

Diagnosis: Prematurity. Dermatitis.

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name H. No. 2. Twin of H. No. 1. Adm. No. 9273

Month ♀ Sex Birth Weight 4 lbs. 8 ozs. Length 19 inches

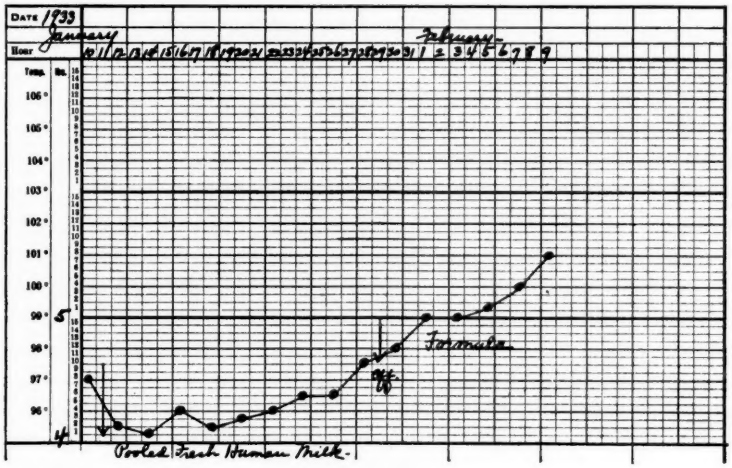

CHART IV.

Premature 48 days.

Birth Weight 4 lbs. 8 oz. Length 19 inches.

Time on Fresh Human Milk (Pooled) 1/11/33—1/29/33 = 18 days.

Increase in Weight 4 lbs. 1 oz.—4 lbs. 11 oz. = 10 oz. Average daily gain .6 oz.

Vitamins added, for 21 days—None.

Refusal—14 times, average 2 drams. Vomiting once. Stools 5—1. Average 4.

Temperature—Stable after 1st week.

Remarks—Took feedings poorly or with gavage first 10 days.

Caloric intake 63 per lb.

Diagnosis: Prematurity. Dermatitis.

part of the time, and fresh milk part of the time. In two instances fresh human milk was given to one of twins and frozen milk to the other. Two babies of about the same degree of prematurity were fed fresh human milk, and it is especially interesting to compare these infants with others of the same age who were fed frozen milk. In still another instance fresh milk was fed to one of two infants, not twins, while the other was given frozen milk, and at the end of about ten days the process was reversed.

The number of days in which the fresh or frozen milk was given by gavage, including the Breck feeder, and also the number of times the milk was refused in part, have been recorded. For the purpose of presenting the results, the series of cases may be divided into three groups; (1) infants who received fresh human milk, (2) infants who received frozen human milk, and (3) a group of infants, selected from groups one and two, who received fresh human milk for one period, and frozen milk for another. Table II shows the figures recorded for each baby, and table III the figures recorded for each group. There is a slight difference between groups I and II in the number of days on which feedings were given by gavage, and also in the number of babies who were not fed by gavage, as compared with those who were. Likewise, there is a slight difference in the refusal of milk,

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name C. Lv. Adm. No. 55222

Month ♀ Sex Birth Weight 4 lbs. 1 oz. Length 15¾ inches

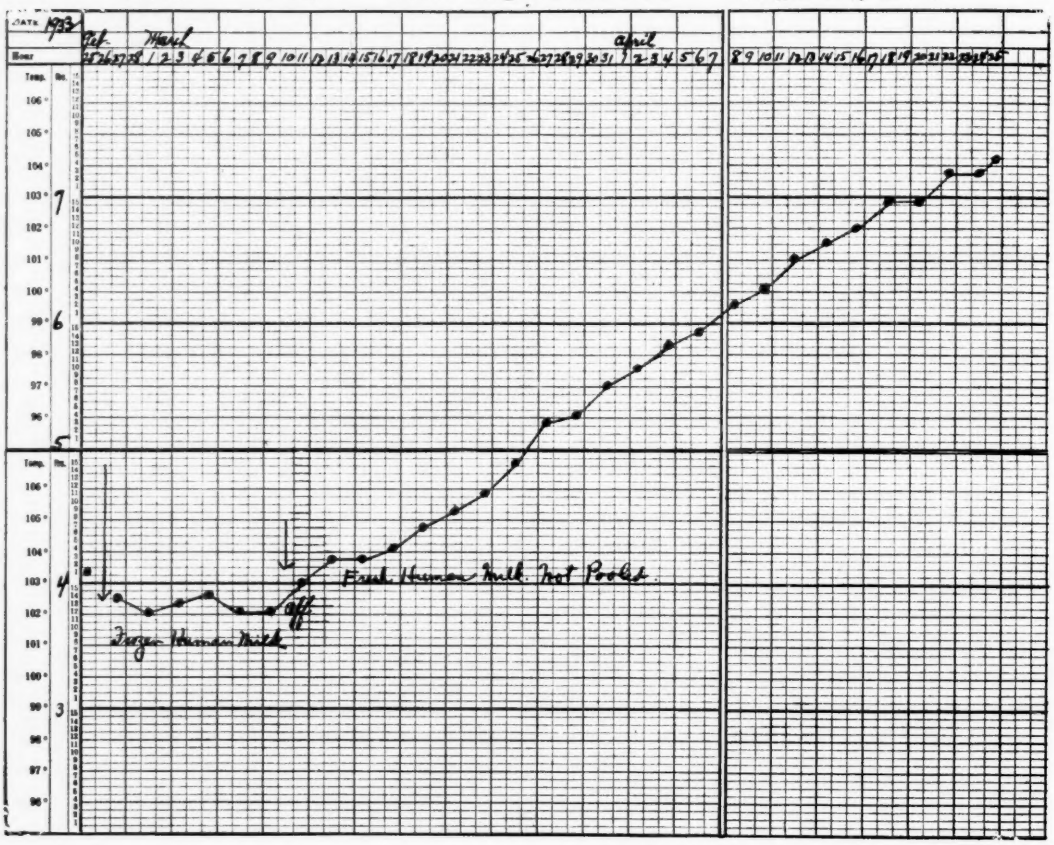

CHART V.

Premature 2 months.

Birth Weight 4 lbs. 1 oz. Length 15¾ inches.

Time on Frozen Human Milk 2/26/33—3/10/33 = 12 days.

Increase in Weight 3 lbs. 12 oz.—4 lbs. = 4 oz. Average daily gain .3 oz.

Age of Frozen Human Milk 72-129 days. Average 113 days.

Refusal—11 times. Vomiting once. Stools 6—2. Average 3+.

Temperature—Stable after 2nd week.

Vitamins added—None.

Caloric intake 66 per lb.

Time on Fresh Human Milk (Mother's) 3/11/33—4/25/33 = 45 days.

Increase in Weight 4 lbs. 4 oz.—7 lbs. 5 oz. = 49 oz. Average daily gain 1.0 oz.

Refusal—5 times. Vomiting once. Stools 6—0. Average 3.

Vitamins added—None.

Caloric intake 70 per lb.

Remarks. Umbilical abscess not found while on Frozen Milk. This baby was fed alternately from feeding to feeding for two days on frozen human milk, and his own mother's milk, without ill effect.

Diagnosis: Prematurity. Umbilical abscess.

INFANTS' HOSPITAL

Name Cov. ♂ Date of Birth No. A 18631

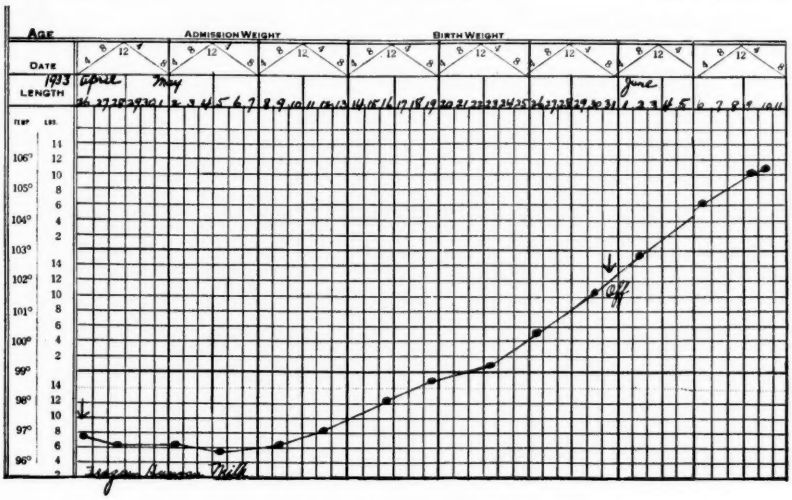

CHART VI.

Premature 2 months.
Birth Weight 3 lbs. 7½ oz. Length 16½ inches.
Time on Frozen Human Milk 4/26/33—5/31/33 = 35 days.
Increase in Weight 3 lbs. 5 oz.—4 lbs. 12 oz. = 23 oz. Average daily gain .7 oz.
Age of Frozen Human Milk 1-220 days. Average 15 days.
Vitamins added—None.
Refusal 0. Gavage 2 weeks. Vomiting 4 times per week first 3 weeks. Stools 4—1. Average less than 2.
Temperature—Stable after 5th. week.
Remarks. Occasional episode of Cyanosis?
Caloric intake 65 per lb.
Diagnosis: Prematurity. Thrush.

INFANTS' HOSPITAL

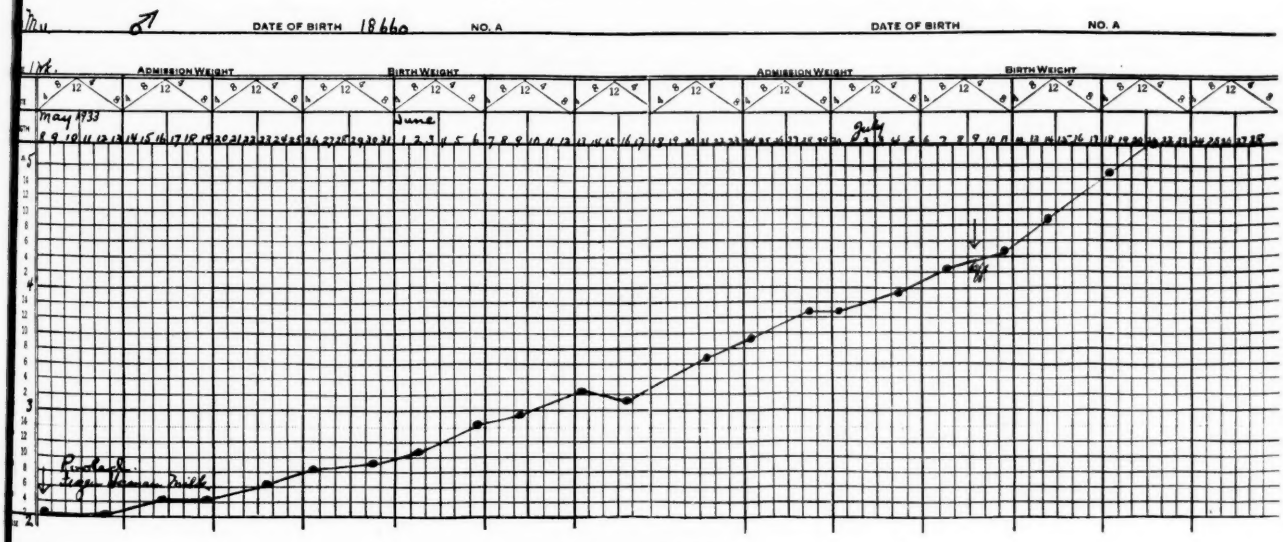

CHART VII.

Premature 1½ months.
Birth Weight 2 lbs. 11 oz. Length 14½ inches.
Time on Frozen Human Milk 5/8/33—7/9/33 = 62 days.
Increase in Weight 2 lbs. 2 oz.—4 lbs. 3 oz. = 33 oz. Average daily gain .5 oz.
Age of Frozen Human Milk 1-120 days. Average 13 days.
Vitamins added, for 2 months—None.
Refusal 0. Gavage 4th, 5th, 6th, 7th week. Vomiting 3 times weekly for 2 weeks. Stools 4—0. Average 1.
Temperature—Unstable first 8 weeks.
Remarks. Cyanotic, limp and dehydrated on entry. Given artificial respiration on the way to the hospital. After entry required clyses and O2 and CO2 Treatments and Adrenalin. Two Transfusions were given for failure to gain.
Caloric intake 60 per lb.
Diagnosis: Prematurity. Double Otitis Media.

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name N. Re. Adm. No. 7275

Month ♀ Sex Birth Weight Length

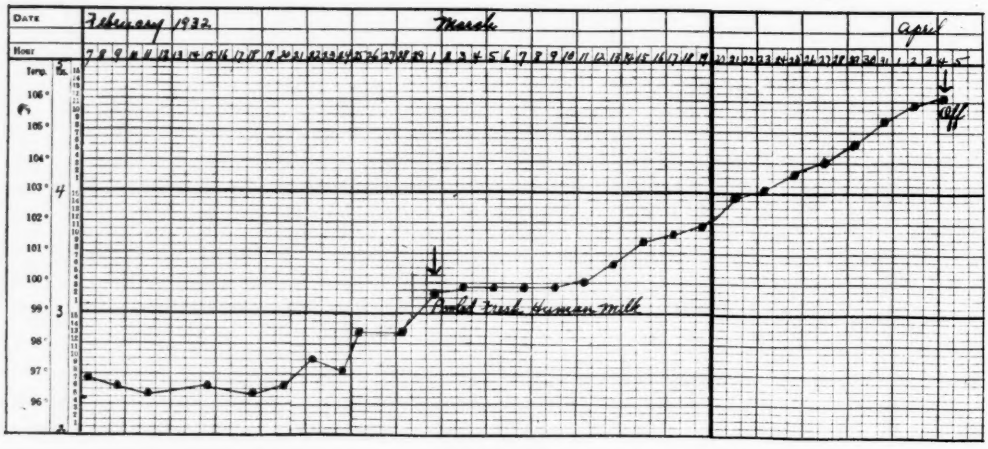

CHART VIII.

Premature 66 days.
Birth Weight 2 lbs. 7 oz. Length 16 inches.
Time on Fresh Human Milk (Pooled) 3/1/32—4/4/32 = 34 days.
Increase in Weight 3 lbs. 4 oz.—4 lbs. 12 oz. = 24 oz. Average daily gain .7 oz.
Refusal—9 times. Gavage and Breck 31 days. Vomiting 13 times, average 3 drams. Stools 5—0. Average 2+.
Temperature—Stable after 4 weeks.
Vitamins added, for first 5 weeks—None.
Remarks. Stormy course for 2 weeks of Vomiting and Cyanosis, requiring O_2 and CO_2 tent, clyses and 2 Transfusions of blood.
Caloric intake 67 per lb.
Diagnosis: Prematurity. Thrush.

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name Le B. Adm. No. 8661

Month ♀ Sex Birth Weight 3 lbs. 5 ozs. Length 19 inches at 7 wks.

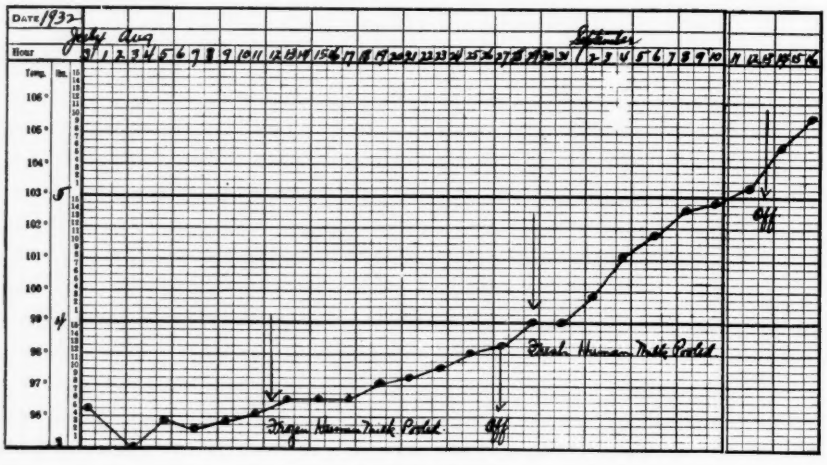

CHART IX.

Premature 75 days.
Birth Weight 3 lbs. 5 oz. Length at birth ? At 7 wks 19 in.
Time on Fresh Human Milk (Pooled) 8/29/32—9/13/32 = 15 days.
Increase in Weight 4 lbs.—5 lbs. 2 oz. = 18 oz. Average daily gain 1.2 oz.
Refusal—5 times, average 2-6 drams. Gavage whole or in part 4 days. Vomiting 2 times, average 1 dram. Stools 6—2. Average 4.
Temperature—Stable after 2 weeks.
Vitamins added, for first 4½ weeks—None.
Caloric intake 57 per lb.
Time on Frozen Human Milk 8/12/32—8/27/32 = 15 days.
Increase in Weight 3 lbs. 5 oz.—3 lbs. 13 oz. = 8 oz. Average daily gain .5 oz.
Age of Frozen Human Milk 22-166 days. Average 82 days.
Refusal—20 times, average 2-8 drams. Gavage 14 days. Vomiting once, average 4 drams. Stools 5—2. Average 4.
Temperature—Stable after 2 weeks.
Caloric intake 63 per lb.
Remarks. Marked Asphyxia the first two days. Given one transfusion of blood 8/27/32 because of apathy and loose stools. General course very satisfactory.
Diagnosis: Prematurity. Asphyxia.

Form 106

BOSTON LYING-IN HOSPITAL

BABY CHART

Babies Ident. No. Unit Hist. No.

Name M. Pa. Adm. No. 700

Month ♀ Sex Birth Weight 3 lbs. Length 16¼ inches

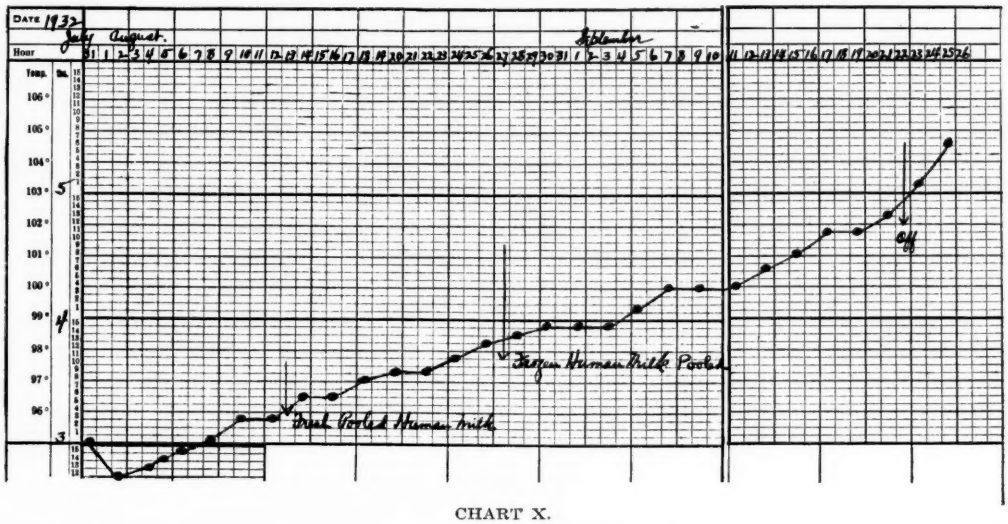

CHART X.

Premature 31 days.
Birth Weight 3 lbs. Length 16¼ inches.
Time on Fresh Pooled Human Milk 8/13/32—8/27/32 = 14 days.
Increase in Weight 3 lbs. 4 oz.—3 lbs. 13 oz. = 9 oz. Average daily gain .6 oz.
Refusal—7 times, average 4 drams. Gavage 3 days. Vomiting once, average 1 dram. Stools 5—2. Average 3+.
Temperature—Stable after 4th week.
Vitamins added, for first 4½ weeks—None.
Caloric intake 63 per lb.
Time on Frozen Human Milk 8/27/32—9/22/32 = 26 days.
Increase in Weight 3 lbs. 13 oz.—4 lbs. 15 oz. = 18 oz. Average daily gain .7 oz.
Age of Frozen Human Milk 1-28 days. Average 15 days.
Refusal—2 times, average 3 drams. Vomiting none. Stools 6—2. Average 4.
Remarks. No cause could be found for the questionable convulsion.
Caloric intake 65 per lb.
Diagnosis: Prematurity. ? 1 Convulsion.

INFANTS' HOSPITAL

Name W. Kn. ♂ Date of Birth No. A 18453

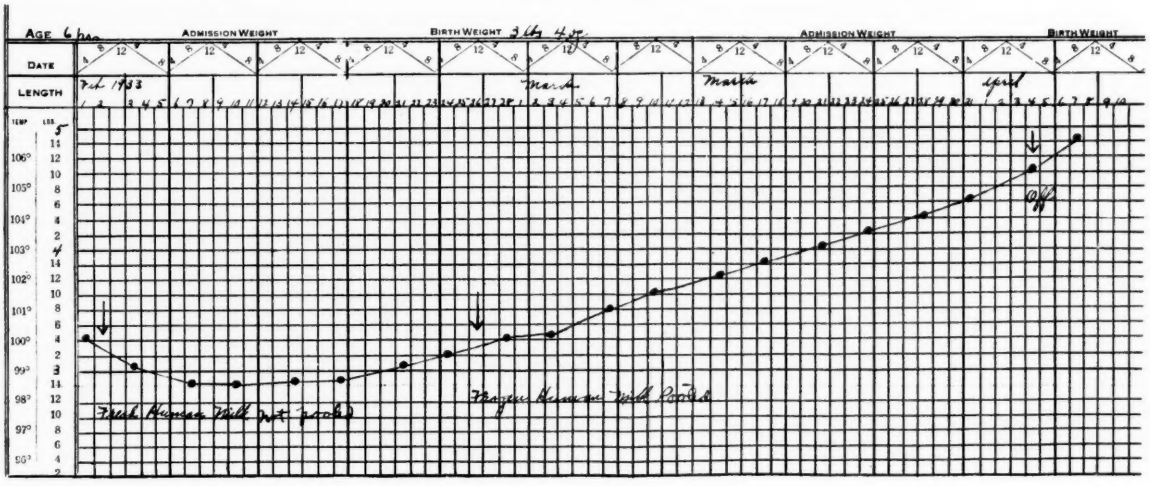

CHART XI.

Premature 1½ months ?
Birth Weight 3 lbs. 4 oz. Length 16 inches.
Time on Fresh Human Milk 2/2/33—2/26/33 = 24 days.
Increase in Weight 2 lbs. 14 oz.—3 lbs. 3 oz. = 5 oz. Average daily gain .2 oz.
Refusal—None. Vomiting 6 times. Stools 7—1. Average 3.
Caloric intake 61 per lb.
Time on Frozen Human Milk 2/26/33—4/4/33 = 37 days.
Increase in Weight 3 lbs. 3 oz.—4 lbs. 10 oz. = 23 oz. Average daily gain .6 oz.
Age of Frozen Human Milk 4-129 days. Average 50 days.
Vitamins added, for first 7 weeks—None.
Refusal—None. Vomiting 6 times. Stools 8—0. Average 3.
Temperature—Unstable for 7 weeks.
Caloric intake 61 per lb.
Diagnosis: Prematurity.

INFANTS' HOSPITAL

Name R. Ha. ♂ Date of Birth No. A 18422

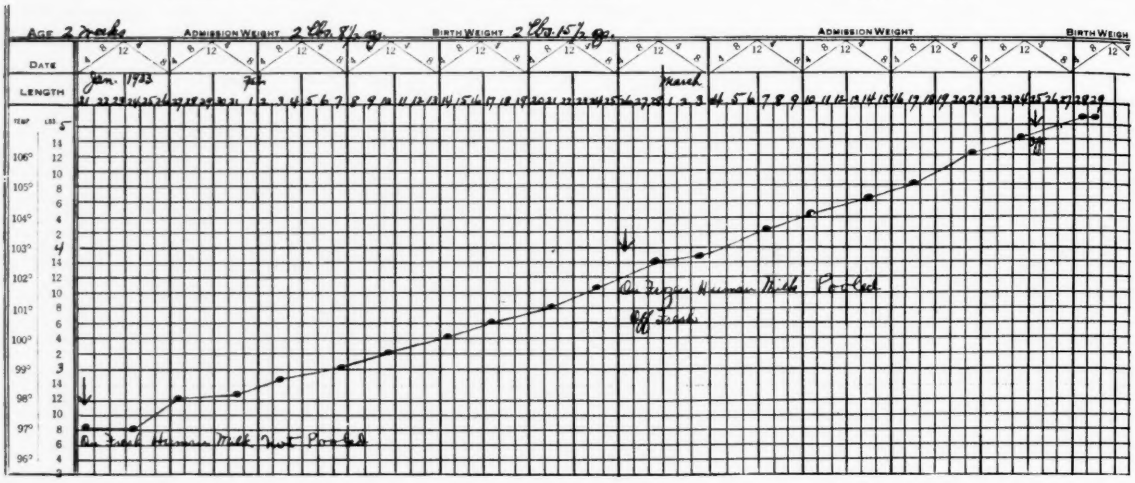

CHART XII.

Premature 2 months.
Birth Weight—2 lbs. 15½ oz.
Weight at entry—2 lbs. 8½ oz.
Length at 5 weeks—16.1 inches.
Time on Frozen Human Milk 2/26/33—3/25/33 = 27 days.
Increase in Weight 3 lbs. 13 oz.—4 lbs. 15 oz. = 18 oz. Average daily gain .7 oz.
Age of Frozen Human Milk 16-129 days. Average 63 days.
Vitamins added, for first 7 weeks—None.
Refusal—None. Vomiting—None. Stools 6—0. Average 2.
Temperature—Stable after 7 weeks.
Caloric intake 61 per lb.
Time on Fresh Human Milk 1/21/33—2/26/33 = 36 days.
Increase in Weight 2 lbs. 8 oz.—3 lbs. 13 oz. = 21 oz. Average daily gain .6 oz.
Refusal—None. Vomiting—None. Stools 6—1. Average 3.
Caloric intake 60 per lb.
Diagnosis: Prematurity.

INFANTS' HOSPITAL

Name C. Wr. ♀ Date of Birth July 29, 1932 No. A 18022
Age 11 days Admission Weight 3 lbs. Birth Weight 3 lbs. 6 ozs.

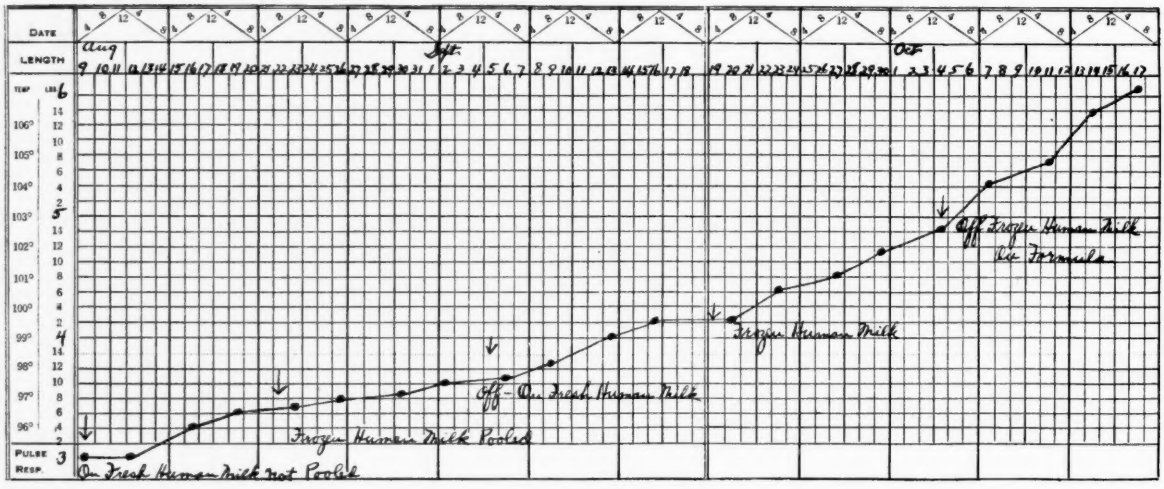

CHART XIII.

Premature 2 months.
Birth Weight ? Length 17½ inches.
Weight at 11 days—3 lbs.
Time on Fresh Human Milk 8/9/32—8/22/32 = 13 days.
Increase in Weight 3 lbs.—3 lbs. 7 oz. = 7 oz. Average daily gain .5 oz. Stools 5—2.
Time on Frozen Human Milk 8/22/32—9/5/32 = 14 days.
Increase in Weight 3 lbs. 7 oz.—3 lbs. 11 oz. = 4 oz. Average daily gain .3 oz. Stools 4—1.
Age of Frozen Human Milk 1-167 days. Average 21 days.
Time on Fresh Human Milk 9/5/32—9/19/32 = 14 days.
Increase in Weight 3 lbs. 11 oz.—4 lbs. 3 oz. = 8 oz. Average daily gain .6 oz. Stools 4—0.
Time on Frozen Human Milk 9/19/32—10/4/32 = 15 days.
Increase in Weight 4 lbs. 3 oz.—4 lbs. 14 oz. = 11 oz. Average daily gain .7 oz. Stools 3—0.
Vitamins added, for first 38 days—None.
Remarks. This baby was apparently moribund on entry and had frequent spells of cyanosis for the first few days. It was transfused the first day, given clyses for 5 days, and gavaged for 2 weeks.
The temperature was stable after the first 2 weeks.

INFANTS' HOSPITAL

Name Fr. ♀ Date of Birth No. A 18516

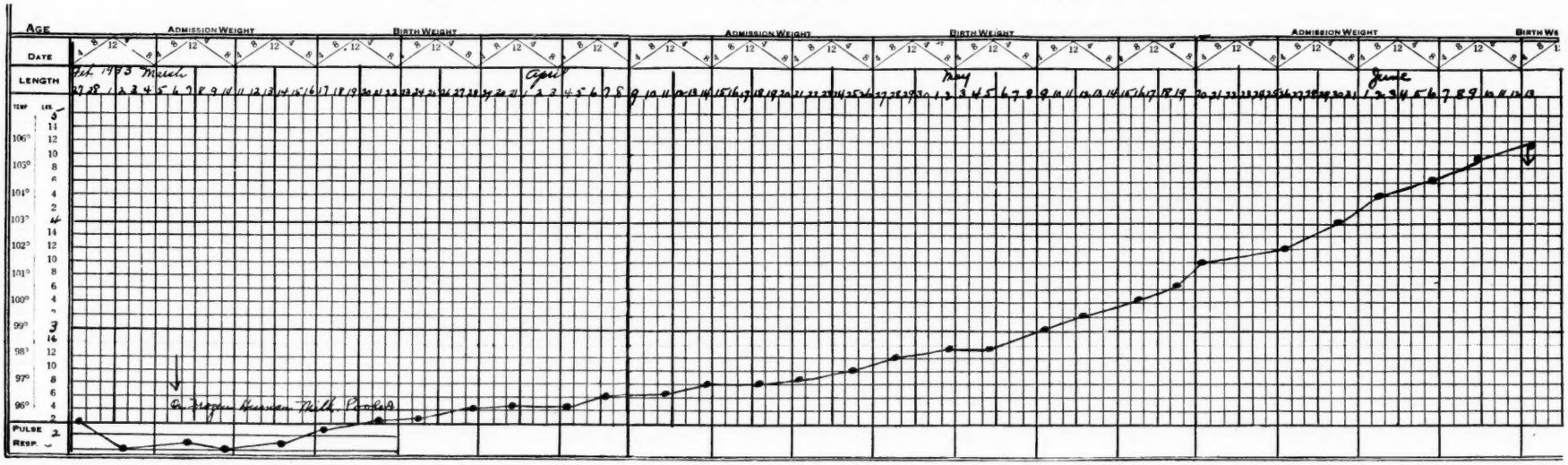

CHART XIV.

Premature 2½ months.
Birth Weight 2 lbs. 2 oz. Length 14 inches.
Time on Frozen Human Milk 3/6/33—6/13/33 = 99 days.
Increase in Weight 1 lb. 15 oz.—4 lbs. 11 oz. = 44 oz.
Average daily gain .4 oz.
Age of Frozen Human Milk 1-220 days. Average 16 days.
Vitamins added, for first 7 weeks—None.

Refusal 0. Gavaged from 2nd to 6th week. Vomiting—average once daily first 4 weeks. Stools 7—0. Average 2.
Temperature—Stable after 5th week.
Remarks. Two transfusions because of loose stools. O2 and CO2 inhalations first five days.
Average Caloric intake 68 per lb.
Diagnosis: Prematurity.

INFANTS' HOSPITAL

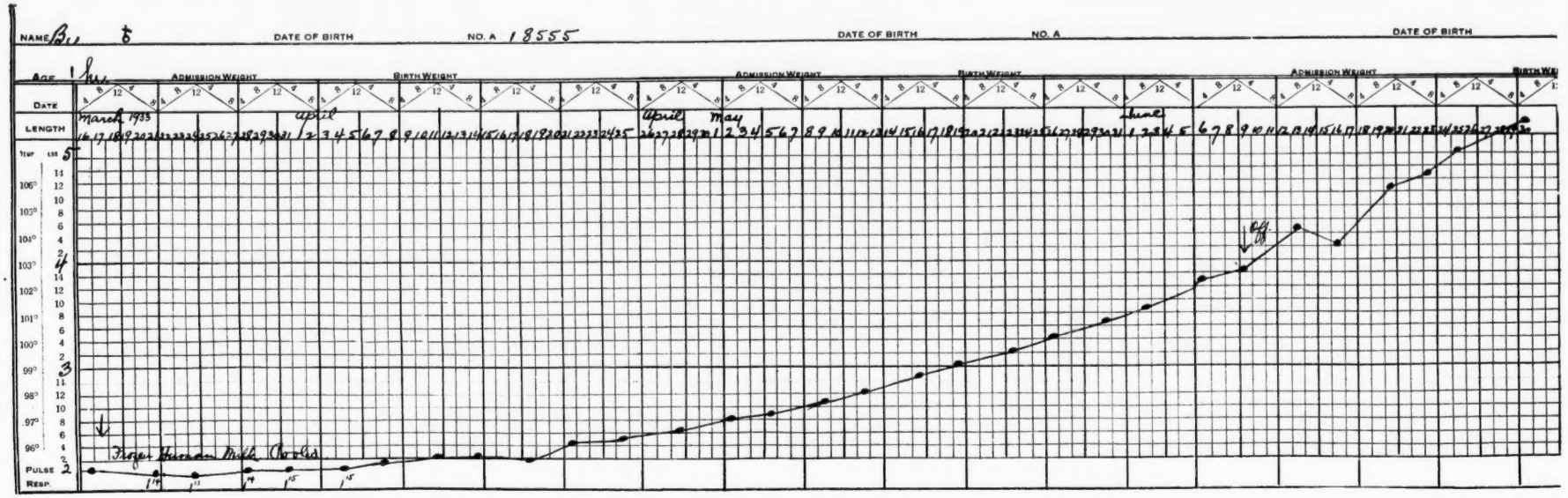

CHART XV.

Premature 3 months.
Birth Weight 2 lbs. Length 14 inches.
Time on Frozen Human Milk 3/17/33—6/9/33 = 84 days.
Increase in Weight 1 lb. 13 oz.—3 lbs. 14 oz. = 33 oz. Average daily gain .4 oz.
Age of Frozen Human Milk 1-220 days. Average 20 days.
Vitamins added, for first 4 weeks—None.

Refusal 0. Gavage 1st, 2nd, 3rd, 5th and 6th weeks. Vomiting 14 times. Stools 7—0. Average 2.
Temperature—Stable at 7th week.
Remarks. 3 Transfusions because of failure to gain. Poor general condition, regurgitation, discharging ear and eye.
Caloric intake 66 calories per lb.
Diagnosis: Prematurity. Atelectasis. Otitis Media.

although the amount refused in any one case was small. In group 3 the number of babies on frozen milk requiring gavage was greater than the number on fresh milk, but a still larger number on frozen milk did not require gavage feeding. The daily record of vomiting and the daily number of stools were recorded. In table III the incidence of vomiting may be seen to be about the same, whether the babies were on fresh or frozen milk. The difference in the number of stools is so small as to be unimportant.

The figures for feeding by gavage, for refusal of food, for vomiting and for the number of stools were obtained also for a series of pairs of babies. T1 and T2 were twins whose courses were similar, the one on frozen milk, the other on fresh milk, (table IV). H2 and H1 were twins, whose courses were similar, (table V). Cov and C Lv (table VI) represent the gain in weight of a pair of babies of almost the same degree of prematurity, the first of whom was fed at the Infants' Hospital on pooled human milk, frozen, the second at the Boston Lying-In Hospital on his own mother's milk, fresh. The difference between them was slight. N.Re and Mu (table VII) were another pair of babies, one fed pooled frozen milk at the Infants' Hospital, the other pooled fresh human milk at the Lying-In Hospital. Their courses were similar. LeB and MPa (table VIII) were babies who were fed on fresh human milk and then on "frozen" milk. The difference is unimportant. These observations show, therefore, that taken by groups, or in pairs, babies fed on frozen human milk seemed to find it as palatable and digestible as fresh milk.

If the amount of weight gained by the babies on fresh milk is compared with that gained by babies on frozen milk, it will be seen, (table II) that as among the three groups, the difference would amount to less than six ounces a month. Taken by pairs, the gain of babies on frozen milk differs very little from the gain of those on fresh milk. (Charts I and II, III and IV, V and VI, VII and VIII, IX and X.) The upward trend, scarcely interrupted, of the weight curve of the babies who were fed first on frozen milk, and then on fresh milk, or vice versa, (charts XI, XII, XIII, XIV and XV), is rather striking. Moreover, the number of required calories varied but little in the several groups of babies, and likewise the time required for a baby to begin to gain was practically the same on frozen as on fresh milk.

In conclusion, we may say that, though we fed premature babies in August on milk that had been frozen in February, they accepted the milk and digested it, as well as they did fresh milk. They gained approximately as much, and they were in excellent condition at the time of discharge.

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5 Smith, L. W., and Emerson, P. W.: Dried human milk. Its use in the feeding of premature infants. Am. J. Dis. Child. 31: 1, 1926.
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Discussion

Chairman O'Keefe: This paper will be discussed by Dr. Charles F. McKhann, Jr., Boston.

Dr. McKhann: It is rather late, and you may wish to get away, but if you have not seen the exhibit of the Directory for Mother's Milk you should visit it.

I think this is a valuable piece of work. All of us who are confronted with the problem of feeding babies artificially are comforted by knowing that there is available a supply of mother's milk. To maintain this supply in the presence of fluctuating demand has been a great problem. For eight years or more, Dr. Emerson, in conjunction with the Directory for Mother's Milk, has sought a satisfactory means of preserving the excess milk which they occasionally have. All of these attempts have been unsatisfactory, or relatively unsatisfactory, until the present one which you have heard described, of freezing the milk. Apparently, reliquefied frozen milk is the equivalent, in so far as we can determine, of fresh milk. The feeding charts which Dr. Emerson has shown you are quite convincing. If you will step over to the exhibit you will see charts of weight curves of babies who are fed alternately with fresh milk and liquefied frozen milk, with apparently no interruption at all in the weight curve.

If further observations confirm these studies that the frozen product is a safe and satisfactory product, and that it can be kept over a period of weeks and months, retaining its natural qualities, it will be a great aid to the Directory because they will have an outlet for their excess collection, and it will mean furthermore that woman's milk can be shipped from Boston to considerable distances. At the present time milk is shipped in the fresh state for some distances, and it seems to me that frozen milk will further aid in this matter.

I think Dr. Emerson and the Borden Company, which has assisted him in this research, as well as the Directory, are to be commended for this undertaking and for the success which has attended it.

Chairman O'Keefe: The paper is now open for general discussion. If there is no further discussion, the meeting will stand adjourned.

VITAMIN D MILK IN THE TREATMENT OF INFANTILE RICKETS*

A Clinical Assay

BY BENJAMIN KRAMER, M.D.,‡ AND ISAAC F. GITTLEMAN, M.D.‡

INTRODUCTORY

THE efforts of clinicians, pathologists, chemists and students of nutrition have thrown much light on every aspect of rickets. Many agents have been developed, which when administered in adequate dosage prevent the disease or cure it after it has developed. So numerous are these that the physician confronted with the task of choosing an antiricketic preparation is confused by their multiplicity. Under these circumstances, such factors as palatability, gastro-intestinal tolerance, dosage, secondary deleterious effects and expense play an important rôle in the choice of the agent to be used. The logical agent is one in which the antiricketic factor is an integral part of the basal diet of the infant, and yet aside from endowing this basal diet with adequate antiricketic potency does not modify it in any other way either as to digestibility, palatability, odor, content in other vitamins, etc. The only antiricketic agent which meets all of these requirements is vitamin D milk that is produced naturally by cows that are fed supplemental rations rich in vitamin D.

Preparations Used

Two forms of vitamin D milk are now available in the open market: 1) milk from cows that are fed irradiated yeast, 2) directly irradiated milk†.

Yeast Milk

Weech[25] was first to show that infants nursed by mothers receiving cod liver oil have a lower incidence of rickets or their rickets is less severe than is the case when the nursing mother fails to receive this supplement. In 1924, Lesné and Vagliano[14] gave cod liver oil to cows and subsequently fed the milk to infants. Golding, Soames and Zilva[6] increased the antiricketic potency of milk by feeding cod liver oil to the lactating animals. Irradiation of nursing mothers was found by Hess[8] and his co-workers to increase the antiricketic potency of their milk while the same was shown by Steenbock and his collaborators[19] to be true of lactating goats but not of cows. This, however, seemed to be less effective than direct irradiation[3]. Gerstenberger[5] found that the milk of wet nurses fed irradiated ergosterol cured rickets in infants. The possibility of enhancing the vitamin D content of milk by feeding irradiated yeast had been investigated by Wachtel[24] in Germany and by Steenbock[18], and his co-workers in this country[13]. In 1930, Thomas and MacLeod[23] showed that when amounts of irradiated yeast containing 100,000 units of vitamin D are fed for several days to cows producing 15-30 kilograms of milk per day the milk acquires a potency of 160 units of vitamin D per liter.

Irradiated Milk

In 1925[12] the senior author succeeded in curing eight infants with severe rickets by feeding to each infant about one liter of milk daily. This milk had been irradiated with the mercury vapor quartz lamp for one hour*. The calcium and inorganic phosphorus concentration of the serum was restored to the normal level and a marked retention of these elements was brought about at the expense of fecal calcium and phosphorus. Shortly before the appearance of this communication, Cowell[2] reported the cure of two children with rickets by means of irradiated milk. These results were promptly confirmed by György[7] and others[26]. Apparatus was devised abroad for the irradiation of milk on a large scale and such milk has been used in institutions and even in small communities[17], but vitamin D milk has only recently become available for general use in this country.

The subsequent introduction of methods for the biological assay of vitamin D made it possible to determine the antiricketic potency in terms of rat unts of irradiated milk as well as of other vitamin D preparations. However, Hess[10] was first to point out that clinical potency and vitamin D content as measured by assay on rats are not necessarily parallel.

The irradiated milk† used in this study was produced by brief exposure of fluid milk in thin layers to the flaming carbon arc lamp, using C carbons[20, 21, 22]. Such an arc emits radiations of 2000-3000 Angstrom units. A total of about 3½ million ergs is allowed to play on each cubic centimeter of milk for a period of 16 seconds. Milk thus prepared contains from 50-60 units of

*From the Pediatric Department of the Jewish Hospital, Brooklyn, N. Y. Aided by a grant from Standard Brands, Inc., New York City.

†For simplicity throughout this article these two types of vitamin D milk will be called "Yeast Milk" and "Irradiated Milk", respectively.

‡Kramer — Associate Professor of Pediatrics, Long Island College of Medicine, Brooklyn. Gittleman — Intern, Jewish Hospital of Brooklyn. For records and addresses of authors see "This Week's Issue," page 920.

*In his original communication he intimated that a shorter period of irradiation would suffice.

†Borden's Vitamin D Milk Irradiated was purchased in Detroit and shipped daily by express to New York City. Bioassays by the Fleischmann Laboratories showed it to have an average potency of 55 Steenbock units per quart.

vitamin D per liter. According to Hess and Lewis, and Supplee and his co-workers such milk suffers only an insignificant loss of vitamin A in the course of irradiation. We, however, did not have an opportunity to check this point.

All of these researches left little doubt that vitamin D either when administered to the lactating mother or animal or when formed in vivo by the maternal organism following irradiation finds its way into the milk. The controversial point was, however, whether this attains a sufficient concentration in milk to be effective when taken by the infants in ordinary amounts. The answer to this question could only be forthcoming as a result of clinical test.

Previous Observations

Hess, Lewis, MacLeod and Thomas[10] investigated the comparative clinical potency of irradiated yeast milk, cod liver oil and viosterol and concluded that the ratio of their effectiveness was as 1:1.25:5.2 in the above order.

In a subsequent study of directly irradiated milk, activated in the same manner as the milk used in our study, Hess and Lewis[11] were able to obtain the same clinical results with 750 cc.-1000 cc. of directly irradiated milk, containing 37-50 units of vitamin D as with yeast milk which assayed 150-160 units. They therefore placed the latter preparation in second place in order of antiricketic potency. However, they did not attempt to determine the minimal effective amount of either vitamin D milk. Well-controlled experiments of Gerstenberger and Horesch[4] on two ricketic children would indicate that a quart of milk containing even 50 units of vitamin D produced by cows fed irradiated ergosterol may suffice to cure rickets when fed daily over a period of four weeks or longer. Although the weight of evidence seems to be in favor of the contention of Hess and Lewis, namely, that 37-50 units of directly irradiated vitamin D fed daily in 750-1000 cc. of milk will protect against rickets or cure the disease, such evidence as these authors present constitutes inadequate proof of their contentions.

Hess and Lewis[11] studied thirty-six babies whose ages ranged from 1½-6 months over a period of three months beginning in January and ending the last week in March of the same year. Each child received 750-1000 cc. of milk daily containing 50 units of vitamin D per liter.

The authors conclude that "of the 36 babies that presented no evidences of rickets in January, all but one remained free from roentgenologic signs of rickets." It is doubtful whether a three-month period is sufficient for such a demonstration, particularly in view of the fact that at the end of the observation period all of these 36 infants presented one or more of the so-called clinical signs of rickets, i.e., beading, craniotabes, or bowing. If we accept the interpretation of Hess and Lewis that these signs constitute evidence of "a very mild form" of the disease it is not unlikely that a longer period of observation would have revealed roentgenologic signs, since even after this short period five patients showed "spreading" and one, definite rickets.

Rickets is certainly not so common as heretofore[16]. Hess, Lewis, MacLeod and Thomas from a clinical survey carried out during the winter of 1930-31 among infants and children attending the New York City Child Health Stations showed that fully half of the white infants and approximately three-fourths of the Negro infants have definite clinical signs of rickets. When only patients showing roentgenologically demonstrable disease are included these figures fall to 15 per cent among the white and 30 per cent among Negro infants. In searching for material for our study only infants showing one or more clinical signs of rickets and subsisting on a diet lacking in antiricketic vitamins were chosen and x-rayed. Most of these infants were of the Negro race. Of sixty-four children studied roentgenologically 17 or 26 per cent showed definite evidence of rickets. Three of these were subsequently shown to be undergoing spontaneous healing. It is unfortunate that Hess and Lewis did not consider it necessary to observe a control group of infants over an equal period of time. In view of the fact that even in Negro infants 70 per cent were found to be free of roentgenological signs of rickets, we must consider this omission a serious one.

In studying the curative value of directly irradiated milk the same authors omitted all controls. Three of our series of 17 patients were found to be undergoing spontaneous healing after three weeks, although both serological and roentgenological evidence indicated the disease to be active on admission. Mitchell, Stokes and their co-workers[15] reported that they were able to prevent and cure infantile rickets with milk containing only 20 rat units of vitamin D per liter. No details are given. This astounding result certainly requires confirmation before it can be accepted. Although it is wholly unlikely that all of the infants studied by Hess and Lewis could possibly have been healing spontaneously, the introduction of a preliminary control period and the simultaneous observation of untreated cases under identical dietetic and environmental conditions would have made the demonstration more convincing. Hess and Lewis conclude, "It is not possible to draw a reliable comparison between the efficacy of activated milk and that of the 'yeast milk' which was used in a similar study a year ago. It is certain, however, that the activated product was at least as efficacious. A more extensive experience, particularly one in which the two types of therapy are carried out at the same time and under similar clinical conditions, will be necessary in order to enable a more exact comparison." We have made such a study.

PROCEDURE

We have assumed that any method which will cure rickets will also prevent it, and have preferred to concentrate our efforts on the treatment of a small group of ricketic infants kept on a controlled diet under controlled conditions of environment.

Our patients were selected from those attending the various milk stations and municipal hospital dispensaries of Brooklyn.* All but three were colored. Their ages ranged from three to 22 months. The children were housed in two large wards, one of which faced an inside court. The windows were closed during the day with the exception of a single transom at the end of the room. The wards were thoroughly ventilated at night. The failure of children to improve until vitamin D milk was given may be cited as evdence that such an amount of sunlight as was admitted did not suffice of itself to produce healing. For the sake of uniformity all the children were given a diet of one quart of milk, four ounces of cooked farina and two ounces of orange juice daily. The amount of food refused was carefully noted. After the required preliminary period was over, the same diet was maintained except that a quart of vitamin D milk was substituted for ordinary milk.

The children were divided into four groups. Group one received one quart of milk containing 55 units of vitamin D yeast milk, group two the same amount of milk containing 40 units of vitamin D from yeast fed cows, group three received one quart of irradiated milk containing 55 units and group four the same amount of milk containing 40 units of vitamin D from irradiated milk. The progress of events was followed by periodic x-rays of all the extremities and analyses of the blood serum for calcium and inorganic phosphorus. Complete physical examinations were done on admission and on discharge and whenever indicated during the period of treatment. Complete quantitative chemical studies of the blood serum were made during the active stage of the disease and when healing was well advanced. Metabolic studies were also carried out. These will be reported in subsequent communications.

For yeast milk we used vitamin D certified milk produced by the Walker-Gordon Laboratories. It assayed by rat test 80 Steenbock units per quart. It was appropriately diluted with ordinary Grade A milk to yield either 55 or 40 Steenbock units to the quart. The irradiated milk assayed 55 Steenbock units to the quart. It was used as such in Group 3 and was diluted with ordinary Grade A milk to yield a 40 unit milk for use in Group 4. Both milks were well taken.

Although the ward was isolated in a part of the hospital remote from the children's wards and strict technique was observed a severe upper respiratory infection developed in one child and affected several of the other children in turn. These developed diarrhea and dehydration of varying severity. In one instance the infection proved fatal and postmortem showed caseation of some of the hilum lymph nodes. Intradermal tuberculin tests were uniformly negative on the remaining children. The unfortunate development of this infection explains the failure of some of the infants to gain much weight during the observation period. That this failure to gain weight did not of itself bring on healing is indicated by the absence of spontaneous healing until treatment was begun. Following recovery the same diet was continued until discharge with gratifying results.

Results—Roentgenological

The effectiveness of treatment was determined by noting the interval between the beginning of treatment and the appearance of the first signs of healing; also when healing appeared to be well advanced or complete. The first sign of healing varied with different patients. In some there was increased condensation at the end of the bone, in others a definite line test appeared while still others showed an exaggeration of the cupping with beginning filling in of the cup shaped metaphysis. In very young infants the bone often also showed a very high degree of osteoporosis so that the cortex appeared linear, the lines running parallel to the axis of the bone, while the usual difference in density between cortex and medulla was no longer discernible. At times the appearance of a new center of ossification constituted the first unequivocal evidence of healing.

Chemical

In every instance the chemical findings in the blood serum indicated active rickets, and improvement in blood chemistry ran parallel with the appearance of new calcium deposition in the x-rays. When healing was roentgenologically well advanced the Ca x P product was well outside the ricketic zone.

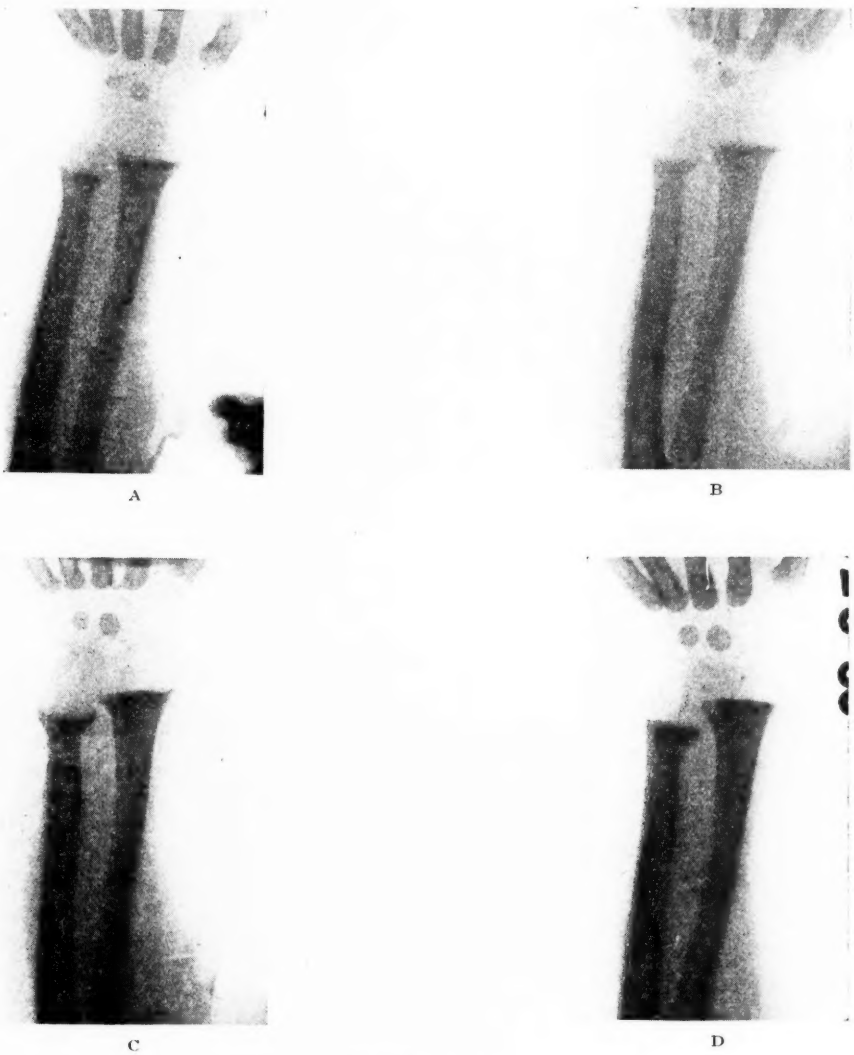
Clinical

All our patients had one or more clinical signs of rickets. However, we did not consider it necessary to continue treatment until all such evidence had been eradicated. Invariably, the children became more active and were obviously subjectively better. Craniotabes disappeared but one or more of the other clinical signs persisted.

*In this work we had the whole-hearted coöperation of Dr. Shirley Wynne, Commissioner of Health of the City of New York, Dr. Herman T. Peck, Deputy Health Commissioner of Brooklyn, and Commissioner Greeff of the Department of Hospitals and of their staffs. It is a pleasure to express our appreciation of their generous and whole-hearted coöperation and to thank the various physicians in charge of the health stations and dispensaries. We appreciate also the permission granted us to bring irradiated milk from Detroit into New York City, without which this investigation would have been impossible.

Cases I, II, III, IV, V

Five infants whose ages ranged from 4-9 months received 55 units of vitamin D in 32 ounces of milk daily. One of the children was white, the rest Negroes. Three of these children were given irradiated milk while the remainder received yeast milk. In each instance the calcium x phosphorus product initially was 40 or less. In one instance (G. W.) where the product was 40 initially, it decreased during the preliminary period of observation. In addition, these patients presented both clinical and roentgenological evidence of rickets. The preliminary period of observation in three children treated with irradiated milk was three weeks in two instances and 20 days in the third instance. The children fed on yeast milk were observed from 18 to 26 days before treatment was begun. In each instance an active process was indicated by the persistence of a low product and the absence of roentgenological evidence of fresh calcium deposition. Every type of case was represented in this group—infants with normal calcium and low inorganic phosphorus, others with low calcium and normal or slightly reduced inorganic phosphorus, while still others had a moderate reduction of both calcium and inorganic phosphorus. In each instance the reduction was sufficient to bring the product within the ricketic zone.



A B C D

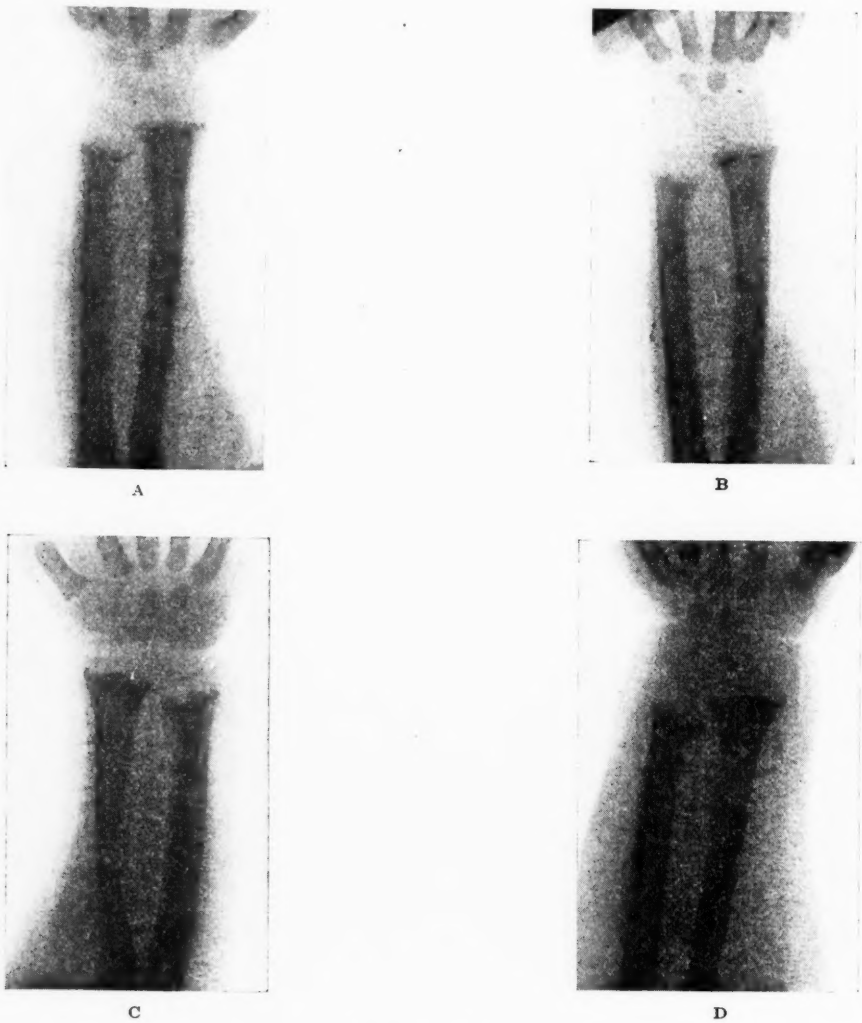
CASE I. J. W.

A—Roentgenogram of right forearm and wrist. 4/5/33—Rickets. Treatment begun 4/26/33.
B—Roentgenogram taken 4/21/33—No change.
C—Roentgenogram showing definite healing. 5/10/33.
D—Healing complete—6/26/33.

When irradiated milk was given the first sign of healing appeared in one case in two weeks and was well marked at the end of 24 days. In a second instance the first signs of healing appeared at the end of 34 days. (It was necessary to discontinue treatment for five days dur-

ing this period). Healing was well advanced in 38 days. The third case showed both a low calcium and a low inorganic phosphorus concentration in the serum, and the early signs of healing were present one week later. Ten days later healing was marked and appeared almost complete one month after treatment was begun.

ing were demonstrated roentgenologically, and a week later this was well marked. Healing was almost complete one month and ten days after the beginning of treatment. The serum calcium and inorganic phosphorus were both normal at this time. The second child treated with 55 units of yeast milk to the quart had what ap-



A B C D

CASE V. L. C.
A—Roentgenogram of right forearm and wrist, initial 3/23/33. Treatment begun 4/19/33.
B—Roentgenogram taken 4/17/33. Rickets more severe.
C—Definite healing 5/12/33.
D—Rickets almost healed 5/20/33.

Two infants were treated with 32 ounces of yeast milk containing 55 units of vitamin D per quart. The first child was a case of mild rickets. During the preliminary period the condition became definitely worse. Serum calcium dropped from 10 to 8.4 mgm. and inorganic phosphorus from 4 to 3.2 mgm. After two weeks of treatment the calcium concentration was normal and inorganic phosphorus had risen to 3.6 mgm. Four days later or 19 days after beginning of treatment the first signs of heal-

Infants with low serum calcium presented no other evidence of tetany. (Electrical reactions were not done but Chvostek and Trousseau phenomena were absent.)

peared to be roentgenologically mild rickets. Blood chemistry, however, showed a calcium concentration of 6.5 and inorganic phosphorus of 4.3. At the end of a 17 day preliminary period the condition seemed to be worse. The first definite signs of healing appeared at the end of 27 days of treatment. Healing was almost complete after one month and six days.

Cases VI, VII, VIII, IX, X.

Five children were given 32 ounces of milk containing 40 units of vitamin D. In three of these the vitamin D was derived from yeast milk

and in the remaining two from irradiated milk. Only one child of this group was white while the rest were Negroes. Their ages ranged from three to 22 months. There were three males and two females. All showed clinical signs of rickets. In all but one case the Ca x P product was either on the border line of rickets or definitely within the ricketic zone.

A B C D

CASE VII. S. A.

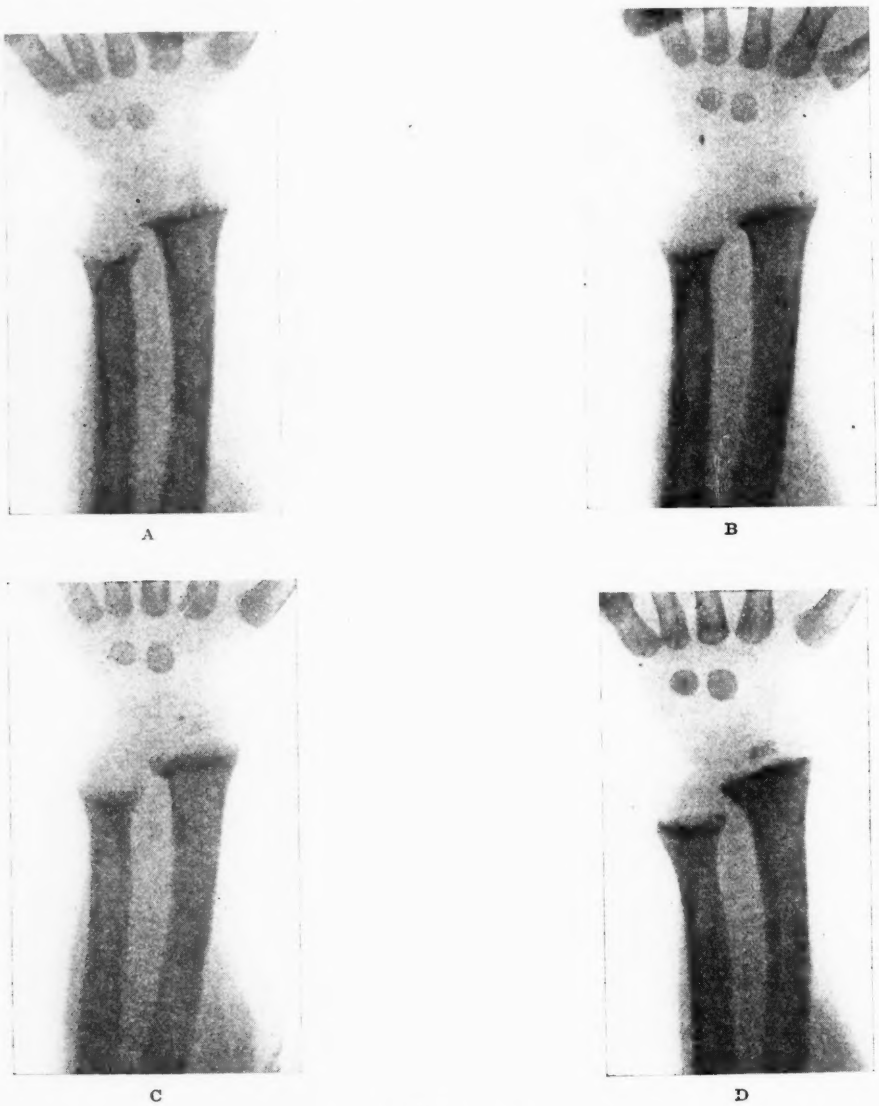
A—Roentgenogram of right forearm and wrist, initial 5/16/33. Treatment begun 5/20/33.
B—Roentgenogram after 14 days of treatment 6/3/33. Beginning healing.
C—Healing advanced 6/27/33.
D—Roentgenogram 7/3/33.

The first child who received 40 units of yeast milk had both early congenital lues and rickets. The chemical findings in the blood showed a normal calcium but a low inorganic phosphorus. A second chemical study nine days later showed no significant changes in the calcium and inorganic phosphorus values. In view of this finding it was decided to treat this patient. Thirteen days later signs of healing appeared and within three weeks after the beginning of treatment the blood calcium and inorganic phosphorus were normal. Healing of the rickets was roentgenologically almost complete within a month. The most striking case was that of S. A., colored female, 22 months of age. This patient was the most severe case of rickets in our entire series. She had been under observation for three weeks in another hospital without treatment. On admission to our hospital she showed severe rickets roentgenologically. The blood serum had a low inorganic phosphorus and a moderate reduction of the calcium concentration. Treatment with 40 units daily of yeast milk was begun on the twentieth of May. Beginning healing was first noted on the third of June, or 14 days later. The calcium and phos-

phorus product had risen to 30. In this instance there was definite evidence of healing although the product was only about 30.

Case VIII, (L. B.) was a colored female infant that had craniotabes, enlarged wrists and a mild rosary on admission. Roentgenological signs of rickets were meagre so that it was difficult to determine the first sign of healing and Nevertheless, even when healing was almost complete the value of the product still did not exceed its initial value. On discharge the serum calcium and inorganic phosphorus values were normal.

The remaining two patients were treated with milk containing 40 units of vitamin D from irradiated milk. One of the patients had a low



A B C D

CASE X. R. F.
A—Roentgenogram of right forearm and wrist, initial. 4/22/33.
B—Treatment begun—5/18/33. Roentgenogram—5/22/33.
C—Earliest x-ray evidence of healing—5/26/33.
D—Advanced healing—6/26/33.

it was therefore necessary to withhold decision until this was well marked. The serum Ca x P product, however, was only 32 due to a low inorganic phosphorus concentration and this did not change after ten days. It is possible that in this case the onset of healing was associated with a transient drop in the Ca x P product.* This was later followed by a rise.

*Kramer and Shear have demonstrated such fluctuation in the product during active healing of experimental rickets in rats.

calcium and normal phosphorus. The other had a normal calcium and low phosphorus in the serum. The first infant was three months old. After a preliminary period of 17 days the x-ray revealed no change in the condition of the bones. Blood calcium decreased nearly a milligram. Treatment was begun on the twenty-third of April. Two weeks later healing was first demonstrable roentgenologically and serum calcium had risen to 8.7 mgm. On the twenty-

sixth of May, one month and three days after the beginning of treatment, healing was almost completed and both the serum calcium and inorganic phosphorus were normal. The second patient went through an attack of pneumonia and was observed for 27 days before treatment was begun. At the end of this period the calcium was 9.6 mgm. and the phosphorus 2.5 mgm. per 100 cc. of serum. X-ray showed no change. Treatment was begun on the eighteenth of May and the first signs of healing appeared eight days later. At this time the serum calcium was 9.5 and phosphorus 3.9 mg. Ten days later the calcium and inorganic phosphorus became normal and healing was well marked.

Controls

The writer is aware of only three studies on vitamin D milk in which adequate controls were used [12, 4, 27]. The importance of such control is illustrated by Cases XI and XII which on admission showed both x-ray and chemical evidence of active rickets while at the end of the preliminary period without intentional treatment presented undoubted evidence of spontaneous healing. The failure of other infants to show healing although observed over the same period as the treated children under identical environmental conditions and receiving a similar diet but without vitamin D adequately disposes, we believe, of the possibility of spontaneous healing in the treated series, due to seasonal or other extraneous factors.

Case XI, R. W., showed a definite increase of calcium and phosphorus in the serum at the end of the preliminary period of 12 days. Unfortunately, no x-rays were taken at this time. Nevertheless, it serves to show that at least in some instances a preliminary period of 12 days is sufficient to demonstrate spontaneous improvement. X-rays taken soon after showed very definite fresh calcification. Case XII, M. B., illustrates the same point. A preliminary control period of two weeks disclosed a definite healing tendency. That the diet which the children received and the environment in which they lived did not suffice to produce healing is shown by Case J. V., XIII, a five months old white child that was observed for a control period of 27 days under the same conditions and upon the same diet as the treated cases. At the end of this period x-rays of the bones remained unaltered and the serum calcium and inorganic phosphorus were both lower than initially. Even a loss of weight which occurred during this period failed to bring on spontaneous healing. Attention should be called to the fact that R. F., Case X. and J. B., Case II, were observed for a preliminary control period lasting from April twenty-second to May eighteenth without showing any tendency to heal, while during this same period the treated infant, L. M. was healing rapidly. Similarly, Case II, J. B. showed no roentgenological improvement between April fifth and May twenty-sixth although during this period seven patients (R. F., L. M., G. W., E. L., J. W., L. C. and B. F.) healed their rickets completely or in part.

DISCUSSION

In determining the potency of any antirickетic agency on rats many factors must be accurately standardized. Obviously, no such exact control is possible in clinical tests. We have attempted to approximate these conditions, however, by placing in each group infants of about the same age and race. The same diet in about equal amounts was given to all and was consumed in toto. The criteria used were the appearance of roentgenological evidence of healing and the time required for healing to be either well advanced or complete. We did not feel it necessary to continue the treatment until all the clinical signs had been eradicated.

Probably the most surprising finding is the cure of rickets with as little as 40 units of vitamin D given along with the basal diet of one quart of milk, cereal, and orange juice. Hess, Lewis and their collaborators[10] failed in several instances to cure rickets with 750 cc. of yeast milk when the cows received 30,000 units of vitamin D per day instead of the maximum of 60,000 units. As they do not give the vitamin D assay of their milk, we have no basis of comparison of our results with theirs. We appreciate that the number of our cases is insufficient to draw any final conclusions as to the relative potency of these two types of vitamin D milk. We can say, however, that the data available do not justify the statement that one milk is less potent than the other.

SUMMARY

Ten children suffering from rickets were treated with vitamin D milk produced either by irradiating the milk directly with the carbon arc lamp or by feeding irradiated yeast to the lactating cattle. The ten infants were divided into four groups, making it possible to feed each of the milks at two levels of vitamin D, that is 55 and 40 Steenbock units respectively per child daily. In each group healing began in from seven days to 31 days, the average period being about two weeks. Healing was well advanced in from four to six weeks. The calcium and inorganic phosphorus concentrations of the serum followed the same course as during treatment with cod-liver oil. Infants showing a low calcium or a low phosphorus or a decrease in the level of both elements in the serum responded equally well to both types of vitamin D milk when these were fed either at a high or low level. Aside from gastro-intestinal disturbances resulting from accidental infection, the milk was well tolerated by all the children.

It is to be regretted that our study was not begun until the early spring and that the period of observation necessarily extended into the ear-

ly summer. We believe, however, that the controls used were sufficient to exclude the possibility of spontaneous healing due to chance irradiation or to foods inadvertently endowed with antirickетic properties. Due to seasonal effect, it would be desirable to repeat these observations during a season of the year when rickets is most likely to be active, and when spontaneous healing is least likely to occur. We hope to be able to continue these studies next winter.

CONCLUSIONS

1. The definite curative action of as little as 40 Steenbock units daily shows vitamin D milk to be our most effective antirickетic agent.
2. At the levels fed there was no significant difference in effectiveness between vitamin D milk prepared by direct irradiation and vitamin D milk produced naturally by feeding cows irradiated yeast.
3. The apparent discrepancy between the minimal clinically effective dose of vitamin D in the form of vitamin D milk and in the form of cod-liver oil or viosterol requires further investigation.

CASE SUMMARIES—YEAST MILK GROUPS 1 AND 2

Case	Treatment	Date	No. Days	X-Ray Diagnosis	Serum mg./100 cc. Ca	Serum mg./100 cc. P	Ca × P
IV. GW.							
4 mos.	None	4/6	0	Mild rickets (upper)			
Male.	(Control)	4/17	11	Rickets (lower)	10.0	4.0	40
Colored.	17 days	4/20	14		8.4	3.2	27
Weight		4/21	15	Rickets worse			
6.78 Kg.							
to	Yeast	4/23	Start				
7.32 Kg.	Milk	5/9	16		9.9	3.6	36
	55 units	5/12	19	Beginning healing			
	daily	5/20	27	Healing more advanced	8.8	4.4	39
		6/3	39	Healing almost complete	10.3	5.8	60
V. LC.							
5½ mos.	None	3/23	0	Mild rickets (upper)			
Female.	(Control)	3/25	2	Rickets (lower)			
White.	17 days	3/29	6		6.5	4.3	28
Weight		4/7	15	Same			
7.2 Kg.							
to	Yeast	4/10	Start				
8.35 Kg.	Milk	4/17	4*	More active			
	55 units	4/20	5*		7.3	3.8	28
	daily	5/12	28	Definite healing			
		5/20	36	Healing almost complete	9.5	6.0	57
VI. BF.							
6 mos.	None	4/10	0	Lues—possible—rickets			
Female.	(Control)	4/12	2	Rickets ?	9.8	3.8	37
Colored.	13 days	4/21	11		9.6	4.0	38
Weight							
6.57 Kg.	Yeast	4/23	Start				
to	Milk	5/6	13	Beginning healing (upper)			
7.7 Kg.	40 units	5/10	17		9.6	5.7	55
	daily	5/12	19	Beginning healing (lower)			
		5/19	26	Advanced healing	9.7	5.6	54
VII. SA.							
22 mos.	None	5/16	13†	Severe rickets			
Female.	(Control)	5/17	14		9.1	3.5	32
Colored.	17 days						
Weight							
9.75 Kg.	Yeast	5/20	Start				
to	Milk	5/27	7	No definite change			
10.8 Kg.	40 units	6/3	17	Beginning healing			
	daily	6/5	19		9.1	3.2	29
		6/19	33	Advanced healing	9.5	4.0	38
		7/3	44	Healing far advanced	10.2	4.8	49
VIII. LB.							
5 mos.	None	4/27	0	Rickets			
Female.	(Control)	5/1	4		10.1	3.2	32
Colored.	16 days	5/11	14	Same	9.1	3.5	32
Weight							
6.9 Kg.	Yeast	5/13	Start				
to	Milk	5/23	10	Same	8.6	3.3	28
7.7 Kg.	40 units	5/31	18	Beginning healing	8.9	1.7	15
	daily	6/2	20		9.8	2.7	26
		6/14	32	Almost healed	9.9	3.5	35
		7/3	51	Healed	10.7	5.7	61

*Yeast milk discontinued 4/14, resumed 4/19 (5 days).
†At Kingston Avenue Hospital, without treatment, from 5/3/33 to 5/16/33 (13 days).

CASE SUMMARIES—IRRADIATED MILK GROUPS 3 AND 4

Case	Treatment	Date	No. Days	X-Ray Diagnosis	Serum mg./100 cc. Ca	Serum mg./100 cc. P	Ca × P
I. JW.							
5 mos.	None	4/5	0	Rickets	8.8	3.3	29
Male.	(Control)	4/12	7	More advanced	9.4	3.0	28
Colored.	21 days	4/21	16	No change			
Weight							
5.88 Kg.	Irradiated	4/26	Start				
to	Milk	5/10	14	Healing beginning			
7.7 Kg.	55 units	5/20	24	Healing advanced	9.5	5.9	56
	daily	6/12			9.8	5.7	56
		6/27	60	Healing about complete			
II. JB.							
9 Mos.	None	4/5	0	Severe rickets	5.8	4.3	25
Male.	(Control)	4/13	8		5.3	4.7	25
Colored.	21 days	4/21	16	No change	5.4	4.6	25
Weight							
7.95 Kg.	Irradiated	4/26	Start				
to	Milk	5/17	15*	No change			
8.52 Kg.	55 units	5/26	24	No change	7.6	4.0	30
	daily	6/2	31	Beginning healing			
	*No D milk	6/5	34		8.9	4.5	40
	5/11 to 5/17	6/27	56	Healing almost complete	10.5	6.0	63
III. LM.							
5 mos.	None	3/23	0	Rickets			
Female.	(Control)	3/29	6		7.9	3.5	28
Colored.	20 days	4/7	17	No change			
Weight							
5.9 Kg.	Irradiated	4/10	Start				
to	Milk	4/17	7	Beginning healing			
6.45 Kg.	55 units	4/27	17	Advanced healing			
	daily	5/2	22		9.8	5.3	52
		5/10	30	Healing almost complete			
IX. EL.							
3 mos.	None	4/5	0	Rickets	7.3	5.7	42
Male.	(Control)	4/20	15		6.3	6.0	38
Colored.	18 days						
Weight							
6.06 Kg.	Irradiated	4/23	Start				
to	Milk	5/6	13	Beginning healing	8.7	6.0	52
6.46 Kg.	40 units	5/20	27	Healing more advanced			
	(daily)	5/23	31		9.9	6.0	59
		5/26	34	Healing almost complete			
X. RF.							
18 mos.	None	4/22	0	Rickets (upper)			
Male.	(Control)	4/24	2		9.6	1.6	15†
Colored.	26 days	4/27	5		9.6	2.5	18
Weight		5/8	16	Rickets			
9.46 Kg.		5/10	18		9.6	2.5	24
to							
10.6 Kg.	Irradiated	5/18	Start				
	Milk	5/22	4	No change			
	40 units	5/26	9	Beginning healing	9.5	3.9	37
	daily	6/9	22	Healing far advanced			
		6/12	25		10.2	5.9	60
		6/26	38	Healing almost complete			

*Irradiated milk was discontinued 5/11 and resumed 5/17 (6 days).

†In the absence of antiricketic therapy the rickets failed to heal although the infant had an attack of pneumonia. Furthermore, the serum phosphorus failed to increase markedly after subsidence of the fever as occurs when antiricketic therapy is given during the acute stage of the pneumonia.

CASE SUMMARIES—UNTREATED CONTROLS

Case	Treatment	Date	No. Days	X-Ray Diagnosis	Serum mg./100 cc. Ca	Serum mg./100 cc. P	Ca × P
XI. RW.							
5 mos.	None	3/29	0	Rickets	9.0	2.7	24
Male.	Discharged	4/10	12		9.7	4.0	39
Colored.	Spontaneous	4/15	17		9.6	4.2	40
Weight	healing	4/17	19	Definite healing			
8.58 Kg.							
XII. MB.							
3 mos.	None	4/27	0	Severe rickets			
Male.	Discharged	5/1	4		9.6	3.0	29
Colored.	Spontaneous	5/10	13	Definite healing	9.5	3.7	35
Weight	healing	5/20	23	Healing advanced	9.6	4.0	38
11.6 Kg.							
to							
12.93 Kg.							
XIII. JV.							
5 mos.	None	3/18	0	Rickets			
Male.	Discharged	3/22	4		7.3	4.0	29
White.	against	3/23	5	No change			
Weight	advice	4/4	17	No change			
7.1 Kg.		4/5	18		7.1	3.6	26
to		4/15	28		6.8	3.5	24
6.24 Kg.							

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19441

A CASE OF DIZZINESS, DOUBLE VISION AND HEMIPLEGIA

NEUROLOGICAL DEPARTMENT

PRESENTATION OF CASE

DR. MARIAN W. ROPES:* This is the case of a forty-seven year old housewife who entered complaining of double vision, dizziness and nausea of three weeks' duration.

The past history was essentially negative until two years before entry, when she had a radical operation on the right breast. The specimen showed malignancy but no apparent involvement of the axillary glands. One year before entry she had a simple operation on the left breast. There was no evidence of malignancy.

Three weeks before admission she began to complain of double vision, most marked on looking to the right, occasionally on looking to the left. During the following week she complained of dizziness and nausea. The symptoms increased until a few days before admission, when she had some difficulty in writing, putting in extra letters and not being able to form some of the letters.

On admission the physical examination was essentially negative except for the neurologic findings. There were nystagmoid jerks on looking to the right, definite nystagmus on looking to the left. There was slight facial weakness on the right apparently involving the whole face. There was slight reduction of hearing on the right. A lumbar puncture showed an initial pressure of 125, seven lymphocytes, total protein 98, colloidal gold 4333322110, sugar 73, Wassermann questionable.

During her stay in the hospital she developed marked headache and became stuporous. During the first week she complained of some weakness of the right side of her face, and the second week showed right hemiplegia. At that time she had bilateral positive Babinski and bilateral ankle clonus. A lumbar puncture at that time showed an initial pressure of 240, total protein 148, cell count five lymphocytes. She became progressively more stuporous and died seven weeks after the onset of the symptoms.

*Assistant resident on the Medical service.

CLINICAL DISCUSSION

DR. ROPES: X-ray had shown evidence of metastases to the chest.

The discharge diagnosis was brain tumor metastasizing from the breast.

DR. AUBREY O. HAMPTON: We have skull films which show nothing and a chest film which does show fairly typical metastases. The right breast has been removed. There is a round nodule one centimeter in diameter in the pleura and a mass here, and probably masses here that do not show quite so well.

DR. TRACY B. MALLORY: If the nodule were not present in the parenchyma of the lung would you be able from the glands to make a diagnosis of metastatic carcinoma?

DR. HAMPTON: No. We could say that the glands were enlarged and the pleura thickened. If we knew the patient had had her breast amputated for carcinoma we would take some odds that it was metastasis.

DR. JAMES B. AYER: Just before she came in I saw this patient with Dr. Stratton of Melrose, who had made a diagnosis of malignancy of the brain, and I was forced to agree with him.

The examination at that time was as Dr. Ropes gives it. Vertigo was the principal symptom, with questionable right facial weakness which developed within a week into a certain facial palsy of the peripheral type. The rest of the findings were as given. It seemed quite certain that we were dealing with a tumor of the brain. We were not perfectly certain as to the localization. About all we were justified in saying was that it probably was a brain stem lesion on the right side.

I think it is interesting that the fluid in this case showed a normal pressure, which is often the case in brain tumor, but with rather a high protein, which is also characteristic of tumor below the tentorium and of acoustic neuroma, which we did not think this was.

The Wassermann bothered us a little, and potassium iodide did not help. Subsequently the Wassermann was negative, and another laboratory checked that, because I think there was nothing else to indicate lues in the case.

As soon as we got the x-rays we did not consider surgery, I think correctly.

An interesting point which seems to be very characteristic of brain metastasis from carcinoma of the breast is duration. There is a patient now in the Baker with almost certain brain metastasis five years following a mastectomy, and the absence of local recurrence is just as striking. In this case there was no local recurrence and nothing in the axilla. In the other patient there is a gland in the axilla.

The possibility of there being more than one tumor is a very real one, and of course the symptomatology here can be explained on the basis of more than one. However, one on the

right side of the pons involving the seventh and eighth nerves can explain everything.

DR. HAMPTON: I should like to say that it is not safe to depend on the chest film alone to rule out metastasis. We know from the work of Dr. Richard Dresser that metastasis from the breast has a fifty-fifty chance of occurring in the pelvis or chest separately, a twenty-five per cent chance in the bones of the skull separately and a forty per cent chance in the vertebrae without either the pelvis or the chest being involved. If we missed finding it in her chest we should have gone on to examine most of her bones separately.

DR. MALLORY: On the other hand a cancer of the breast in order to metastasize to the brain must pass through the lung. It is perfectly possible to do that without producing a visible lesion in the lung, but in the majority of cases we should be able to find a definite focus of growth in the lung. In this case it was very small. The x-ray I think has magnified it a little. We measured it as only seven millimeters in diameter.

CLINICAL DIAGNOSIS

Brain tumor, metastatic from the breast.

ANATOMIC DIAGNOSES

(Carcinoma of the breast).
Metastatic carcinoma to the lungs, the bronchial glands and the brain.
Bronchopneumonia.
Chronic thyroiditis (Hashimoto's struma).
Operative wounds, old: appendectomy, right radical mastectomy, left simple mastectomy.

PATHOLOGIC DISCUSSION

DR. MALLORY: There was a large amount of tumor in the bronchial lymph nodes, evidently secondary to this little focus in the periphery of the lung. The tumor in the brain was nearly four centimeters in diameter and lay largely in the right lobe of the cerebellum near the cerebellopontine angle. The seventh, eighth, ninth and tenth nerves were in immediate apposition and may well have been damaged by direct pressure.

DR. HAMPTON: Was the pleura over the right lung involved?

DR. MALLORY: I think it was not, that is not by the carcinoma.

DR. HAMPTON: We always miss it. I wondered if it was covered.

DR. MALLORY: No, the pleura was negative.

DR. AYER: Dr. Channing Simmons, who removed the tumors of both breasts, said the one on the right was malignant, but was removed as early as one can possibly make the diagnosis. The operation was thorough, and he felt that there could not have been a better chance for recovery. The tumor which was removed a year before entry was definitely benign. So the brain metastasis seems to go back to the malignant tumor of two years previously.

CASE 19442
A CASE OF ESOPHAGEAL OBSTRUCTION

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. ALLEN M. BOYDEN:* This is the case of a sixty-four year old Hebrew who entered the hospital with the chief complaint of difficulty in swallowing of three months' duration.

His story goes back to one year before entry, when he began to notice eructation of gas which bothered him considerably and increased. He was troubled with no other symptoms until eight months before entry, when he noticed some burning in the neck just above the sternum. Three months before he entered the hospital he noticed difficulty in swallowing solid food. This gradually increased in a period of a few weeks and he began to regurgitate a small amount of solid food after he had swallowed it. This symptom increased until about two months before entry, when he began to have difficulty in taking liquids. The day before entry he was unable to take anything by mouth. He had no symptoms of any acute abdominal discomfort previous to entry. He had lost forty pounds during the year and twenty during the past three months.

He was seen in the Eye and Ear Infirmary two weeks before entry. At that time an esophagoscope was passed almost to the cardia. It showed a few granulations there, but no outcroppings sufficient for biopsy. He was referred to the wards.

Physical examination on entry showed a thin, pale man of sixty-four. The examination was not particularly remarkable. His heart was not enlarged. He had a systolic murmur heard over the whole precordium, most marked at the apex. The blood pressure was 95/60. His trachea was not displaced. There were no palpable lymph nodes. His abdomen on examination revealed some tenderness in the right side, but no masses and no spasm were made out. Rectal examination was negative. He had a swallow of barium after entry and showed a definite obstruction in the lower portion of the esophagus.

Shortly after his entry operation was performed. Following operation I did not see him. His temperature chart is lost, and I have not been able to get in touch with the men who did see him. The records of the laboratory work, including the blood count, were lost with the temperature chart. He died three days after operation.

CLINICAL DISCUSSION

DR. BOYDEN: At operation the stomach was found to be markedly involved by an annular growth involving part of the cardia and extending down over the major portion of the stomach, so that it was very difficult to free the stomach. A gastrostomy was performed. A

*Interne on the West Surgical service.

catheter was placed towards the duodenum by the usual technic and the abdomen closed. No metastases were found in the liver or peritoneum.

CLINICAL DIAGNOSES

Carcinoma of the esophagus.
Bronchopneumonia.

ANATOMIC DIAGNOSES

Epidermoid carcinoma, grade II, of the stomach or esophagus with constriction of the esophageal opening.
Operative wound, gastrostomy.
Appendiceal abscess.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The autopsy showed a very large carcinoma, obviously utterly inoperable, which was apparently in the stomach rather than in the esophagus, but so high up in the stomach that it completely surrounded the cardiac orifice and in one or two areas had grown along beneath the submucosa beyond the sphincter so that there was a slight amount of tumor actually above the cardia. I feel distinctly uncertain as to where it was primary. The gross appearance certainly suggested stomach, but the histology, which shows an epidermoid carcinoma, is more in favor of an esophageal origin.

I think perhaps the main interest in the case lies in the actual mode of death. A very large proportion of the cases in which gastrostomy is done wind up on the postmortem table very shortly with a variety of terminal conditions. This one happens to be a new one. It was a ruptured acute appendix. How long he had had it, whether he had it before the stomach was operated on or not I do not know; but there was a hole over a centimeter in diameter at the base of the appendix and the appendiceal abscess must have contained 500 cubic centimeters of pus. I think we cannot connect it with the operation and must consider it a coincidence.

In retrospect, going back over the history, do you think he had the appendicitis when he came in?

DR. BOYDEN: He must have had appendicitis on entry. However, there was no complaint of abdominal pain and there were no acute attacks of vomiting.

DR. MALLORY: It is really a very small percentage of cases of acute appendicitis that are missed at the present time so far as the pathologist can judge, and he has a pretty good chance. This one was certainly a complete miss.

DR. EDWARD D. CHURCHILL: May I say a word about the advisability of surgery in malignant disease of the thoracic esophagus? Attempts to resect lesions in the upper two thirds of the thoracic esophagus have with one or two exceptions ended disastrously. Personally, I should advise such an attempt only in a very early case in an otherwise healthy young person, and then only with the frank understanding with the patient that life was being staked in a desperate gamble. Surgery has more to offer in case the lesion is in the lower third of the esophagus, and the closer to the cardia the better the chances. Under such circumstances an abdominal exploration should be the first stage. What appears to be a very small lesion of the lower end of the esophagus may turn out to be extensive disease of the cardiac end of the stomach with extension upward along the esophagus. If the stomach is found free of disease, then a special type of gastrostomy may be performed and at the same time the cardiac end of the stomach may be mobilized and its vascular supply interrupted. At a second stage an extrapleural approach is made by resecting the vertebral ends of the ninth to eleventh ribs. The diaphragm is sectioned and the stomach drawn upward into a retropleural position. Resection of the esophageal growth and anastomosis of the esophagus to the stomach may then be accomplished. I really feel that this is a feasible and not unduly hazardous operation for an occasional favorable case.

A PHYSICIAN: How much can be resected with anastomosis?

DR. CHURCHILL: It is impossible to state with certainty; I should estimate four or five inches.

DR. AUBREY O. HAMPTON: These patients do not get much relief from x-ray and radium therapy.

DR. MALLORY: One other possible point in connection with the first case as well as this is that cardiospasm has been frequently proved to be secondary to a lesion in the stomach and in at least some of these cases abdominal exploration and the finding of ulcer or benign tumor of the stomach, when it has been possible to remove that, has relieved the cardiospasm. It is worth bearing that in mind in certain cases of cardiospasm.

DR. HAROLD L. HIGGINS: What are the other causes of complications in gastrostomy?

DR. MALLORY: The majority of patients appear to develop peritonitis and die or to have a terminal bronchopneumonia in a few days. Gastrostomy is not done except in cases of patients who are pretty nearly moribund.

DR. HIGGINS: We do it occasionally in children with esophageal stricture following the taking of lye, and have not observed such complications.

DR. MALLORY: I think it would probably be fair to say that very few of these cases of carcinoma of the stomach with metastases survive very long.

DR. ARTHUR W. ALLEN: Dr. Herman at the Eye and Ear Infirmary looked up this subject and found that the average length of life in carcinoma of the esophagus after the diagnosis was made was three months, as I recall his figures. The expectation of life is surprisingly short. That total includes a tremendous number of patients who die following gastrostomy because they are in such a poor state of nutrition at the time of operation.

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"CLINICAL SCIENCE"

FOR twenty-four years one of the best scientific publications on a special subject, *Heart*, has been published under the direction of a distinguished board of editors in London. Much of the material in it came as a result of the stimulation given to research by the Medical Research Council of England, so ably directed by the late Sir Walter Fletcher. The chief design of the journal was to present the problems of cardiology and for its editor Sir Thomas Lewis, a man of world-wide reputation, was wisely chosen. During the period of its publication, this journal has fulfilled the purpose for which it was founded, even beyond the scope intended by its original sponsors.

In the course of time, however, it has been felt that a journal of such worth should have a wider field than cardiology and now, in 1933, the old journal, *Heart*, has been abandoned and a new journal takes its place, *Clinical Science incorporating Heart**, with Sir Thomas Lewis still as editor. The first number, which has recently appeared, is sufficient guaranty of the excellence of this publication, and the medical profession will welcome a journal of its type, dealing in general with the broad subject of medicine and in particular with clinical research based on the application of physiological principles to the study of hospital patients. A large amount of material in the first issue comes from the Medical Unit of the University College, London, and it is hoped that this famous institution will continue to produce and publish research of the same high standard.

Rarely does the editor of a medical journal feel called upon to draw attention in the editorial columns to a new scientific publication. In the case of *Clinical Science*, however, one feels entirely justified in using this means, for the new journal is sure to be one of great importance to medicine in general.

**Clinical Science incorporating Heart*, edited by Thomas Lewis. Shaw & Sons, Ltd., London. Vol. 1, No. 1, 1933.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CATTELL, RICHARD B. A.B., M.D. Harvard University Medical School 1925. F.A.C.S. Surgeon, Lahey Clinic, Boston. Assistant Surgeon, New England Deaconess and New England Baptist Hospitals. His subject is "Thyroid Disorders in Childhood." Page 867. Address: 605 Commonwealth Avenue, Boston.

BONIN, CLEOPHAS P. D.M.D. Harvard Dental School 1915. Member of Orthodontia Teaching Staff, Harvard Dental School. Orthodontia Staff, Forsyth Dental Infirmary, Boston. His subject is "What the Pediatrician Can Do for Dental Cripples." Page 875. Address: 230 Beacon Street, Boston.

WYMAN, EDWIN T. M.D. Tufts College Medical School 1911. Instructor in Pediatrics, Harvard Medical School. Visiting Physician, Infants' Hospital, Boston. Assistant Physician, Children's Hospital, Boston. His subject is "Vitamin D Milk." Page 889. Address: 319 Longwood Avenue, Boston.

EMERSON, PAUL W. A.B., M.D. Harvard University Medical School 1911. Instructor in Pediatrics, Harvard Medical School. Assistant Visiting Physician, Children's Hospital. Medical Director, New England Home for Little Wanderers. Director of Research, Directory for Mothers' Milk, Inc. His subject is "The Preservation of Human Milk. VII. The Feeding to Premature Babies of Human Milk Preserved by Freezing." Page 893. Address: 319 Longwood Avenue, Boston.

KRAMER, BENJAMIN. M.S., M.D. University and Bellevue Hospital Medical College 1909. Assistant Professor of Physiology, State University of Iowa Medical School. Instructor, Associate and Associate Professor of Pediatrics, Johns Hopkins Medical School. Associate Professor of Pediatrics and Professor of Clinical Pediatrics, Long Island College of Medicine, Brooklyn. Address: Jewish Hospital of Brooklyn, Brooklyn, N. Y. Associated with him is

GITTLEMAN, ISAAC F. M.D. Long Island College of Medicine, Brooklyn, 1930. Intern, Jewish Hospital of Brooklyn. Address: Jewish Hospital of Brooklyn, Brooklyn, N. Y. Their subject is "Vitamin D Milk in the Treatment of Infantile Rickets." Page 906.

MISCELLANY

A TESTIMONIAL DINNER TO DR. GEORGE H. BIGELOW AND DR. HENRY D. CHADWICK

Four hundred and fifty friends and admirers of Dr. George H. Bigelow, Commissioner of Public Health from 1925 to 1933, and Dr. Henry D. Chadwick, newly appointed Commissioner of Public Health, attended the Testimonial Dinner in their honor at the Copley-Plaza Hotel, Boston, on Thursday, October 26.

Originally planned as a farewell to Dr. Bigelow, it was at his suggestion made also a welcome home dinner for Dr. Chadwick, who was for twenty years Superintendent at the Westfield State Sanatorium and who left Massachusetts to serve as Controller of Tuberculosis in Detroit, Michigan, since 1929.

Dr. Frederick T. Lord, President of the Massachusetts Tuberculosis League, was Chairman of the Dinner Executive Committee. He called the meeting to order and after an invocation by Rev. Father George P. O'Conor, Rector of St. Mary's Church, Dedham, turned the gavel over to Dr. John W. Bartol, President of the Boston Health League, who acted as Toastmaster.

General Daniel Needham, State Commissioner of Public Safety, represented Governor Joseph B. Ely, who was unable to be present on account of official business. General Needham, who was a classmate of Dr. Bigelow at Harvard, paid a tribute to his efficiency as a public servant.

Dr. Frederic A. Washburn, whom Dr. Bigelow will succeed as Director of the Massachusetts General Hospital in February, outlined the magnitude of the task that Dr. Bigelow faces when he takes over the direction of the Hospital.

Dr. Charles F. Wilinsky, Director of Beth Israel Hospital and Deputy Commissioner of Public Health of Boston, brought the greetings of the City to Drs. Bigelow and Chadwick in the absence of Mayor James M. Curley.

Mrs. Carl Illig, Chairman of the Public Health Committee of the Federation of Women's Clubs in the United States, told of the immense satisfaction which the women of the State had in coöperating with Dr. Bigelow during the past eight years for the advancement of public health measures. She pledged their support to Dr. Chadwick in his new position as Commissioner.

Dr. Kendall Emerson, Managing Director of the National Tuberculosis Association and Acting Director of the American Public Health Association, expressed the appreciation of public health workers throughout the United States for contributions of Drs. Bigelow and Chadwick. As a New Englander he pointed with pride to the accomplishments of the honored guests of the evening through which Massachusetts has been kept in the forefront in public health work throughout the United States.

Dr. William H. Robey, President of the Massachusetts Medical Society, called attention to the fact that the Department of Public Health was established in 1869 only after the persistent efforts of the organized medical group in pressing the desirability of such an organization home to the General Court. He paid tribute to the efficiency and leadership of Dr. Bigelow and welcomed Dr. Chadwick back to Massachusetts to take up the work as Commissioner of Public Health.

Dr. Roger I. Lee, a member of the Public Health Council, stressed the fact that whatever position Dr. Bigelow may occupy he will carry on his work with energy, imagination and courage. He predicted the building up of even a greater usefulness for the Massachusetts General Hospital under the régime of Dr. Bigelow. On behalf of the Public Health Council, Dr. Lee welcomed Dr. Chadwick to his new position.

Responding to the tributes, Dr. Chadwick expressed his deep satisfaction at being called back by Governor Ely to direct the public health work in the Commonwealth. He told of his experiences in Michigan as Controller of Tuberculosis and President of the Michigan Tuberculosis Association. He expressed the opinion that his experiences in the Middle West since 1929 gave him a new perspective which will be valuable in his work here. Dr. Chadwick read some interesting excerpts from the first report of Dr. Henry Ingersoll Bowditch, first Commissioner of Public Health of the Commonwealth, indicating that the ambitions of the first Commissioner for the Department were most comprehensive. He said that he would in every way possible maintain the advanced standards set by Dr. Bigelow.

Dr. Bigelow was the last speaker of the evening. He expressed his deep appreciation of the kind things that had been said about him and the work of the Department of Public Health but he said that the credit was due not so much to himself as to the loyal colleagues in the organization. Dr. Bigelow's peroration will long be remembered by those who heard it. He stressed the fact that public health undertakings can be successful only to the extent that they are understood and supported by an informed public opinion. This calls

for education not only of those immediately concerned with health matters, but of the public at large in a better understanding of the significance of health in the whole scheme of human betterment.

Among those attending the dinner were Dr. John Ferrell, President of the Rockefeller Foundation of New York; Dr. William F. Snow of New York, Managing Director of the American Social Hygiene Association; Mr. Barry Smith and Dr. Clarence C. Scamman of the Commonwealth Fund of New York; Dr. Leroy M. S. Miner, Dean of Harvard Dental School; Dr. Walter P. Bowers, Editor of the *New England Journal of Medicine;* Dr. Francis P. Denny, Health Officer of Brookline; Professor Curtis M. Hilliard of Simmons College; and Mrs. Leslie B. Cutler, Chairman of the Board of Health of Needham.

The Executive Committee for the dinner in addition to Dr. Frederick T. Lord was composed of the following: Dr. William H. Robey, Dr. Gaylord W. Anderson, Dr. Walter P. Bowers, Dr. Charles F. Wilinsky, Mr. Horace Morison, Professor Curtis M. Hilliard, Hon. Herbert Parsons and Frank Kiernan.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending October 7, indicate a mortality rate of 9.8 and the same rate appears for the corresponding week of last year. The highest rate (15.8) appears for Columbus, Ohio, and the lowest (4.3) for Yonkers, N. Y. The highest infant mortality rate (12.9) appears for Knoxville, Tenn., and the lowest for Des Moines, Iowa, Duluth, Minn., Long Beach, Calif., Lynn, Mass., San Diego, Calif., Trenton, N. J., and Yonkers, N. Y., which reported no infant mortality.

The annual rate for 85 cities is 10.9 for the forty weeks of 1933, as against a rate of 11.1 for the corresponding period of the previous year.

SUMMARY OF DEATHS AND DEATHRATES (ANNUAL BASIS) FROM AUTOMOBILE ACCIDENTS PER 100,000 ESTIMATED POPULATION FOR 86 CITIES FOR CORRESPONDING PERIODS OF 1933 AND 1932

	Week ending		First 40 weeks	
	Oct. 7, 1933	Oct. 8, 1932	1933	1932
Total deaths	175	174	5,861	5,832
Deathrate	24.4	24.7	20.4	20.7
Deaths due to accidents in city	128	121	4,597	4,689
Deathrate	17.8	17.2	16.0	16.7

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR SEPTEMBER, 1933

Anterior poliomyelitis decreased remarkably during the month as was expected from its early July prevalence.

Diphtheria and typhoid fever are running constantly lower than ever before in their history.

Tuberculosis, pulmonary and other forms, encephalitis, epidemic cerebrospinal meningitis, measles, German measles, and chicken pox are all lower than for September of 1932.

The incidence of dog bites was slightly higher than usual, with the occurrence of a human death from rabies.

Scarlet fever continues at a lower level than for the corresponding month of the two preceding years.

Lobar pneumonia, while higher than September of last year, is below the five year average.

Whooping cough continues to show an increased prevalence for the year.

RARE DISEASES

Anterior poliomyelitis was reported from Amherst, 1; Andover, 1; Arlington, 2; Barnstable, 1; Beverly, 3; Boston, 14; Braintree, 1; Brockton, 2; Brookline, 1; Cambridge, 1; Chelsea, 1; Chicopee, 1; Danvers, 1; Fall River, 1; Falmouth, 3; Fitchburg, 1; Georgetown, 1; Haverhill, 1; Hingham, 1; Holyoke, 1; Lawrence, 1; Lynn, 1; Malden, 2; Mansfield, 1; Marlboro, 1; Medford, 1; Methuen, 3; Milton, 1; Natick, 2; Newton, 2; Northbridge, 1; Quincy, 4; Revere, 2; Salem, 1; Saugus, 1; Somerville, 2; Springfield, 2; Stockbridge, 1; Stoneham, 2; Sudbury, 1; Watertown, 2; Winchester, 1; Winthrop, 2; Worcester, 6; out-of-state, 1; Total, 83.

Dysentery was reported from Cambridge, 1; Chelsea, 1; Danvers, 2; Taunton, 1; Total, 5.

Epidemic cerebrospinal meningitis was reported from Middleboro, 1.

Malaria was reported from Boston, 1; Duxbury, 1; Fitchburg, 1; Northampton, 1; Total, 4.

Septic sore throat was reported from Beverly, 1; Boston, 6; Lowell, 1; Lynn, 1; Total, 9.

Tetanus was reported from Brockton, 1; Holyoke, 1; North Attleboro, 1; Taunton, 2; Total, 5.

Trachoma was reported from Boston, 2.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
MONTHLY REPORT FOR SEPTEMBER, 1933

Disease	Sept. 1933	Sept. 1932	Average*
Anterior Poliomyelitis	83	12	169
Chicken Pox	81	81	97
Diphtheria	58	70	152
Dog Bite	576	505	506
Epidemic Cerebrospinal Meningitis	1	7	7
German Measles	10	34	25
Gonorrhea	648	587	523
Lobar Pneumonia	82	67	92
Measles	80	94	116
Mumps	98	111	93
Paratyphoid Fever	3	—	—
Rabies	1	—	—
Scarlet Fever	292	363	311
Syphilis	362	369	235

*Average number of cases for September during the preceding five years.

Tuberculosis, Pulmonary	263	291	312
Tuberculosis, Other Forms	32	34	47
Typhoid Fever	23	30	37
Whooping Cough	500	303	409

CORRESPONDENCE

THE EVILS OF THE "SALOON"

45 Bay State Road,
October 17, 1933.

Editor, *New England Journal of Medicine,*

Much interest is being shown by various groups of people who are especially interested in the several forms of legislation now being proposed for the control of liquor in this state, following the repeal of the Eighteenth Amendment. I believe it is the hope of all our people that whatever form such legislation will take, its aim will be the safeguarding of the interests of all concerned. Yet there is a danger looming high above the horizon, and which may have its roots in a selfishly interested group of people, that threatens to restore to its old-time glory the old-time saloon. Of course, it may be labeled with a high-sounding or romantic name in order to disguise it, but it will be the old-time bar-room and it will function in the same old-time way. That danger is close upon us and unless action in concerted form and by influential forces is taken at once the saloon will be here soon and remain with us as a permanent institution and with all its old-time evils.

A saloon is a saloon by any other name. Let us not be fooled by such romantic names as the "tavern" or "beer-gardens," or by any such custom as is practiced in some countries, that of serving with each drink the inevitable dried-up, miniature cheese sandwich, in order that the law which requires that food be served with liquor be complied with. Nobody who voted for the repeal of the Eighteenth Amendment wants the return of the saloon. Everybody who voted, and those who did not vote, object to the return of the saloon to dot our streets and disgrace our neighborhoods. Why then should such a menace be suggested? It is obvious that somebody is more interested in revenue than in the welfare of the people.

The saloon should not be permitted to return, and every physician should oppose its return, in any of its varied forms, which in reality are nothing more than saloons.

I have had an opportunity to serve on the house staffs of the Foxborough State Hospital and the Norfolk State Hospital when those institutions were devoted to the care and treatment of alcoholics and drug addicts. At one time I was Admitting Physician to the out-patient department of these hospitals, located in Boston. My association with those institutions brought me in intimate contact with alcoholics of all grades and varieties and I have learned from them many of the evils of the saloon. I need not recount them. They are still fresh in the memories of the clinician, the clergyman, the social worker, the jail warden and the police. The saloon made its impress upon those men who frequented it regularly, and the consequences of that sordid association were visited with damaging force upon their unfortunate families.

Many a mild drinker came to the beginning of his downfall, and met the end of his career, in the saloon. I have seen thousands of these human wrecks brought to the hospital, restored to as good mental and physical health as possible, returned to their families to begin again, only to be brought back to us, victims of the corner saloon. These men were not the real sufferers; the real victims were their families. No form of protective legislation will safeguard the wives and children of the saloon victims so long as the bar-room is open. Let us not be fooled into believing that the new type of bar-room will be different from the old type saloon. The effect on the susceptible drinker will be the same, whether he is served in a saloon or a tavern, and after the establishment of this type of drinking place the Commonwealth will be obliged to maintain an institution similar to the Foxborough and Norfolk hospitals of yesterday, for the treatment of inebriety.

Should we stand by placidly watching our legislature return the saloon to us, or should we, as physicians who appreciate the effect of uncontrolled drinking upon the human body, raise our voices in protest?

Let us not forget that the saloon, more than any other factor, has shaped the opinions of those who voted for prohibition, but who have voted against prohibition on the understanding that the saloon would not come back.

I am a strong anti-prohibitionist, but I am a stronger anti-saloonist. Experience with victims of the saloon during the saloon era was my teacher.

Very truly yours,
SOLOMON H. RUBIN, M.D.

REMINISCENCES AND INTERPRETATIONS

Editor, *New England Journal of Medicine,*

When I was a lad, the old cow used to come home at night a bit restive and out of sorts. Perhaps she had had a hard day; the feed had been poor; she had been scratched with brambles and pestered with about a million flies. In tying her up, we had to be wary and soothe her a bit. Your editorial of September 21, "The Care of Persons 'on Relief,'" makes me think of those days, for the last half of that editorial could be condensed into two words: "So, boss!"

And I am indebted to you for many chuckles, for behind the folds of the editorial toga I could see your real self; that you do not think any better of this relief business than we do, but with a big, blue eagle sticking up in front of you,

of course you had to straddle. The "bogey" of State Medicine is a real person up here. And moreover, he has married a wife, and she rules the old man and tells the rest of us where to get off. Inasmuch as most of us already have a wife apiece, more or less, this smacks of double taxation, and you know that New Englanders just never could stand that.

There is another humorous side: When the Relief plan was released, informing doctors that they could collect perhaps an average of ten per cent for the services that they had been in the habit of rendering for nothing, the medical men said, "Fine!" and rose up in high glee. Within ten days they sent in bills enough to swamp the treasury of the United States, which gave rise to many "So, boss!" editorials and warnings from state headquarters. Perhaps it may serve a good purpose in reminding the people that the doctors can still truly say, "We do our part."

LOUIS W. FLANDERS, M.D.

Dover, New Hampshire,
Sept. 27, 1933.

OBITUARIES

RESOLUTIONS ON THE DEATH OF DR. WILLIAM T. COUNCILMAN*

Dr. Councilman's death on May 26, 1933 came in the wane of a long life filled with many satisfactions in service to ideals, in creative and recreative fields, and in affectionate family life. In appreciation of his services to the medical school, to medical science and to medical education, the Medical Faculty of Harvard University records his death with sorrow, the deeper because of the affection he inspired.

Dr. Councilman was born January 1, 1854, in Pikesville, Maryland, on land held by his family since early in the eighteenth century when his great-great-grandfather, Christopher Councilman, came from Holland. His father was a graduate of Yale College and the Maryland University Medical School. His mother was Christiana Drummond Mitchell, of English and Scotch descent.

His knowledge and love of horticulture and gardening were inspired in his early years by his parents. He attended St. Johns College, Annapolis. He studied medicine at the University of Maryland and received his M.D. degree in 1878. Contact with Henry Newell Martin, Professor of Biology at Johns Hopkins University, awakened his interest in research, which he began while assistant quarantine officer of Baltimore. In 1880 he went abroad and spent two years studying with Chiari in Vienna, with Cohnheim and Weigert in Leipzig and with von Recklinghausen in Strassburg. In 1886 he joined Welch at the Johns Hopkins Hospital as associate in pathology, and after another year abroad in 1888 he became assistant professor of pathology and resident pathologist at the Johns Hopkins Hospital. In 1892 he was called to Harvard as the third Shattuck professor of pathological anatomy. Appointed that year visiting pathologist to the Boston City Hospital, he organized the department there and drew Frank B. Mallory into pathology. He induced the Massachusetts General Hospital to secure a building for pathology and to engage, in 1896, a full-time pathologist and recommended James Homer Wright for the position. He fostered the development of a separate department for neuropathology. In 1913 he enthusiastically assumed the duties of the pathological department of the Peter Bent Brigham Hospital. In the American Medical Association he was a member of the Council on Medical Education from 1904 to 1909 and a member of the board of trustees from 1909 to 1917.

His incumbency of the professorship of pathologicay anatomy at Harvard was characterized by vigorous activity and great enthusiasm in teaching and research. He drew many able men into the department who subsequently attained important positions in pathology and medicine here and in other universities. Kindly, tolerant, and observant, his appraisal of men was rapid and accurate. He gave his friendship freely and practised the amenities of life, unadorned by formalities or artificialities. His personality was transparent, for he cultivated neither reserve nor mannerisms: he achieved dignity unconsciously by virtue of directness, competence and honesty. In his pupils he inspired respect and admiration and in highest degree lasting affection.

His duties as administrator and teacher in the medical school and the hospitals were constantly heavy. His most important services were through his influence on teaching and by giving impetus to the development of men of professional calibre. In teaching he stressed learning to observe and independent thinking rather than the acquisition of facts.

His list of publications is not long, though he made numerous important and lasting contributions to pathological literature.

In 1916 he accompanied Dr. A. Hamilton Rice's expedition to the Amazon as medical observer. He returned impaired in health and thereafter suffered from attacks of angina, which in no way seemed to depress him or diminish his enthusiasms over his favorite interests and hobbies. In 1923, accompanied by Mrs. Councilman he went to Pekin as temporary professor of pathology at Union Medical College for the academic year of 1923-24. He resigned his professorship at Harvard in 1923, becoming emeritus. He belonged to several foreign and American medical societies, was a Fellow of the American Academy of Arts and Sciences, Fellow of the Philosophical Society of Philadelphia and member of the National Academy of Sciences.

His honorary degrees were A.M., Harvard Uni-

*Read at the first Faculty Meeting of the academic year, Harvard Medical School, October 6, 1933.

versity, 1899; A.M., Johns Hopkins University, 1902; LL.D., University of Maryland, 1907; LL.D., McGill University, 1911; LL.D., Western Reserve University, 1929; LL.D., St. Johns College, 1932.

He received a gold medal from the National Institute of Social Sciences.

The honors which came his way, the successes of his pupils and their abiding affection for him, reflect the usefulness of his life and the value of his services through the medium of the Medical School.

S. B. WOLBACH,
H. A. CHRISTIAN,
W. B. CANNON.

DONALD JOHN ARMOUR

The death of Mr. Donald Armour in London removes from the medical world one of a group of surgeons particularly interested in the surgery of the nervous system. Neurosurgery, which has grown to such great proportions in this country, in recent years, largely due to the stimulating efforts of Dr. Cushing, has had a somewhat slower growth in England. The pioneer was Sir Victor Horsley, who did his first major neurosurgical operation in 1888. Following Horsley, in London, were Sir Charles A. Ballance, Sir Percy Sargent, and Donald Armour. Sir Percy's death occurred earlier this year. After 1906, Sargent and Armour shared the work at the National Hospital, Queen's Square, where Horsley had done his original neurosurgery. Neither limited himself entirely to this field and it is, perhaps, on account of this that others have made greater contributions to the subject. Nevertheless, the work of both added important data to neurosurgery and their efforts will not soon be forgotten.

Official biographies of Armour will, naturally, appear in the British and Canadian medical journals. Intimate details about the man, however, are sometimes more important in estimating his character than a critical list of his scientific contributions. The author of this note well remembers an incident that occurred in September, 1932. While staying at a hotel in London with Dr. Harvey Cushing, Armour happened to come in and see us. His hospitality was overwhelming, for he not only arranged for us to go to the theatre, that evening, but learning that we were anxious to see Sir Charles Sherrington before leaving England, he planned to take us down to a summer resort on the south coast of England, where Sherrington was staying. The next day he did so, driving his own car, and as we traveled through the fine countryside south of London, he pointed out many historical sites, particularly the famous Knolle House, the home of Lord Sackville-West, recently so well described in the novel, "The Edwardians." We found Sir Charles and Lady Sherrington at Bexhill-on-Sea, where we had a delightful and profitable visit. Arriving back in London late that evening, Armour arranged a pleasant supper, thus completing a day long to be remembered. This type of spontaneous hospitality, in which the host guides himself entirely by the wishes of his guests, is one rarely experienced and leaves a pleasant impression of a man who saw life in its broadest aspects.

RECENT DEATHS

DREW—FRED DREW, M.D., of 259 Beacon Street, Boston, died October 22, 1933, at his home. He was born in Alfred, Maine, in 1866, the son of Ira T. and Lydia A. (Straw) Drew, and graduated from the Harvard Medical School in 1894, after premedical studies at Bowdoin College. He joined the Massachusetts Medical Society in 1895, and again in 1921. He is survived by his widow, Mrs. Adelaide A. (Brown) Drew, and a daughter, Mrs. Corinna A. Procter of Boston.

JONES—LYMAN ASA JONES, M.D., a retired practitioner, died at the Norwood Hospital, October 26, 1933, after a long illness.

Dr. Jones was born in Appleton, Wisconsin, in 1865, and acquired his premedical education at Lawrence College. He graduated in medicine from the Harvard University Medical School in 1891. After studying in Germany, he served as intern at the Worcester City Hospital, and later as assistant physician at the Worcester State Hospital for the Insane. He afterward engaged in private practice in North Adams, Massachusetts, where he held the office of State Health Inspector. He served in several departments of the State Department of Public Health until he was appointed superintendent of the Pondville State Cancer Hospital in 1927 where he served until he retired two years ago. He joined the Massachusetts Medical Society in 1892. He is survived by his widow, Mary Davis Jones, and two daughters, Mrs. M. W. Evans of Chicago, and Miss Helen A. Jones of Walpole.

QUACKENBOSS—ALEXANDER QUACKENBOSS, M.D., died at his home, Trinity Court, Boston, October 27, 1933, after a long illness. He was born in Worcester, Massachusetts, in 1866, and after graduation from Dartmouth College entered the Harvard University Medical School, graduating therefrom in 1892. He served an internship at the Boston City Hospital subsequently joining the staff of the Eye and Ear Infirmary of the Massachusetts General Hospital. He became a recognized authority in his specialty, and was appointed to the position of Professor of Ophthalmology in the Harvard Medical School. He served in the World War with the Harvard Unit of the British forces, and retired with the rank of Major in the medical division. He joined the Massachusetts Medical Society in 1890, and retired in 1931. Dr. Quackenboss is survived by his widow, Sophia Delano Quackenboss.

NOTICES

CORRECTION

In the Massachusetts General Hospital invitation to physicians to attend Staff Meetings which appeared on page 812, issue of October 19, there was a slight error in the schedule. Ward rounds which are scheduled from 8:00 A.M.-9:00 A.M. start in Ward F instead of Ward I as previously stated.

REMOVALS

JONAS R. KAGAN, M.D., announces the removal of his office from 5 Esmond Street, Dorchester, to 25 Columbia Road, Dorchester.

WILLIAM D. MCFEE, M.D., announces the removal of his office to 41 Bay State Road, Boston, on November 1.

H. H. SERUNIAN, M.D., announces the removal of his office to 23 Chandler Street, Worcester, Mass.

UNITED STATES CIVIL SERVICE EXAMINATION

Junior Medical Officer (Interne), $2,000 a Year
Saint Elizabeths Hospital, Washington, D. C.

Applications must be on file with the manager of the Fourth U. S. Civil Service District, Washington, D. C., not later than November 15, 1933.

RADIO HEALTH MESSAGES

NOVEMBER-DECEMBER, 1933

HEALTH MESSAGES

Sponsorship: Public Education Committee of the Massachusetts Medical Society and Massachusetts Department of Public Health.

Courtesy WBZ. Fridays, 4:30 P.M.

Nov.
- 3 Danger Signals in the Rheumatic Infections of Childhood
- 10 Communicable Diseases in the Schools
- 17 Illness and Character
- 24 Heart Disease in Middle Age

Dec.
- 1 The Cause and Treatment of Crossed Eyes
- 8 Infantile Paralysis
- 15 The Preschool Child
- 22 Cancer of the Intestinal Tract
- 29 Preventive Medicine from Your Family Physician

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 1:15 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 5:00 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

HARVARD MEDICAL SCHOOL

THE GEORGE W. GAY LECTURE on "Medical Ethics." Amphitheater C at 5 P.M.

Thursday, November 2—"The Theology of Medicine." Dr. Robert B. Osgood of Boston.

LECTURES ON "THE CARE OF THE PATIENT." Amphitheater C at 5 P.M.

Thursday, November 9 — Dr. John Homans of Boston.

Thursday, November 16—Dr. Elliott P. Joslin of Boston.

THE LAWRENCE CANCER CLINIC

Lawrence, Massachusetts, October 26, 1933.

To the Physicians of the Northern Half of Essex County:

Dear Doctor:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, upon Tuesday, November 7, at 10 A.M., will be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Associate in Surgery at Harvard University Medical School, Acting Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, and Member Cancer Commission of Harvard University, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

Committee

ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D.,
Chairman.

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

On Thursday, November 9, in the amphitheatre of the Peter Bent Brigham Hospital at 3:30 P.M., there will be a clinic conducted by Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, to which physicians are cordially invited.

REPORTS AND NOTICES OF MEETINGS

THE NEW ENGLAND MEDICAL SOCIETY

The one hundred and nineteenth meeting of the New England Medical Society was held in the

Evans Auditorium of the Massachusetts Memorial Hospitals on Thursday, October 19, 1933, at 8 P.M. Dr. L. W. Crockett presided.

The speaker of the evening was Dr. Eugene Pool, Clinical Professor of Surgery at Cornell University Medical College, who presented a paper on "Chronic Duodenal Stasis." The patient with this condition is usually first seen by the internist who frequently makes the diagnosis of neurasthenia, failing entirely to recognize the condition principally because it is not borne in mind. According to Dr. Pool, chronic duodenal stasis is a clinical entity subdivided into three main groups: those cases in which stasis occurs from distortion or compression of the second portion of the duodenum by bands and ligaments, especially the hepatoduodenal; those in which there is a temporary loss of tone in the duodenum, with impaired function; and those cases with obstruction along the course of the duodenum especially at the duodenojejunal juncture. The causes of obstruction are multiple. Pressure from the mesenteric vessels, folds of mesentery, and angulation of the duodenum are common causes.

Among the common symptoms may be mentioned a marked loss of weight accompanied by a progressive asthenia over a rather long period of time. Indigestion and vomiting often leading to tetany are important symptoms. An interesting feature of the symptomatology is the periodicity of the complaints determined apparently by the patient's emotional state. Fever, headache, neuralgia, and skin eruptions are often encountered. If untreated, the patients tend to become seriously neurasthenic.

The diagnosis of the condition, which is made by x-ray, was discussed at some length. The importance of fluoroscopy in both upright and prone positions was emphasized.

The treatment of the condition has been quite conservative. Dr. Pool pointed out that it is neither necessary nor wise to operate on every patient. Operation is undertaken only when intensive medical treatment has failed. Early diagnosis is essential for the success of medical treatment. The longer the history the less the likelihood of cure without surgical intervention. Medical treatment includes a low residue diet of high caloric and vitamine content, prohibition of alcohol and tobacco, and a definite rest program. Atropine and insulin have been tried with some benefit. Surgery is indicated in young patients, in uncontrollable indigestion and vomiting, with marked stasis, and with progressive loss of weight and strength accompanied by psychoneurosis. The operation of choice is duodenojejunostomy. It was indicated in only a third of the cases observed. It is important "to bear the condition in mind, recognize its significance, and suggest proper treatment".

Dr. Pool then reviewed ten of his cases and illustrated them with lantern slides showing points of interest in diagnosis and surgical technique. The striking thing in many of the cases was the multiplicity of operations before the condition was recognized and properly treated. Many of the cases had been followed for several years with no return of symptoms.

The discussion was opened by Dr. David Cheever. Obstruction of the duodenum in man was attributed at least in part to his assumption of the upright posture. It is surprising that obstruction does not occur more frequently. Dr. Cheever pointed out that of 45,000 admissions at the Peter Bent Brigham Hospital only two were classed as dilated duodenum with duodenal stasis. He had operated on both of the cases without benefit in spite of successful duodenojejunostomy, the operation recommended by Dr. Pool. Dr. Cheever warned against popularizing and exploiting the operation as a cure for various ill-defined ailments. He even raised the question whether duodenal stasis may not be physiological as is stasis from angulation of the splenic flexure which serves a useful purpose in absorption.

The discussion was continued by Drs. Charles T. Howard, Fred B. Lund, Richard H. Miller, Irving J. Walker, E. A. Codman, and one of the x-ray men present. The rarity of the condition was emphasized by all. Each of the speakers expressed his lack of familiarity with the subject under discussion. That the condition is one to be always borne in mind, however, was generally recognized. The general surgeon was warned against operation in the neurotic ptotic individual. In no cases should surgical procedure be attempted unless one can be definitely certain of beneficial result from the operation. From the x-ray point of view, it was emphasized that obstruction from nervous imbalance in the neurasthenic ptotic patient has to be carefully ruled out by study of the function of the duodenum.

Dr. Pool closed the discussion by answering a number of questions that had been asked. He emphasized that even when the condition is found, surgery is to be the last resort. He explained the rarity of the condition as being due at least in part to failure of clinicians to recognize it.

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The New England Roentgen Ray Society held its October meeting, in the Evans Memorial Building, 82 East Concord Street, Boston, on Friday, October 20, 1933, at 8:00 P.M.

The X-Ray Department of the Evans Memorial was open for inspection before the meeting. The demonstration of an automatic-control apparatus for predetermining the phase of x-ray exposure in the cardiac cycle, and of other interesting features was well attended and well received.

During the scientific session, Helen Mathews, M.S., presented a short illustrated discussion of some useful x-ray accessories, all of which were developed in the research laboratories of the Massachusetts Memorial Hospitals.

A very instructive paper on "X-Ray Features of Cranial Dysplasias of Pituitary Origin" was read by Dr. George Levene for Dr. Hector Mortimer, who was unavoidably absent. Changes in the functional state of the pituitary cause alterations of the skeleton, especially of the skull. Dr. Mortimer notes four main types, each of which has characteristic x-ray appearances.

Richard J. Wagner, Ph.D., next described and demonstrated a simplified apparatus for the quantitative measurement of x-rays.

In an interesting paper on "Structural Relationships in Abnormal Individuals," Allan W. Rowe, Ph.D., concluded that x-ray studies confirm the validity of weight predictions based on the standards of Dreyer and of Life Insurance Tables.

Dr. Frank E. Wheatley brought out in his illustrated talk many practical points in diagnosis, from roentgenological observation of cholecystitis.

A preliminary report was presented by Samuel H. Caldwell, Sc.D., on an "Automatic-Control Apparatus for Predetermining the Phase of X-Ray Exposure in the Cardiac Cycle." He showed that by amplification of E.K.G. impulses and the adjustment of their time, x-ray pictures could be obtained at any point in the cardiac cycle. Such precision applied to the making of x-ray films of the heart should go a great way in the study and diagnosis of heart diseases by means of the roentgen ray. It is certainly the next important step after the development of impulse radiography.

The final paper was that of Dr. George Levene on "Roentgenological Diagnosis of Coronary Disease." In addition to presenting the x-ray characteristics of the heart in coronary disease, Dr. Levene very graphically illustrated, with ingenious mechanical models faithful in reproduction, the fluoroscopic appearances obtainable in nine of the more important heart conditions, namely, extra systoles, sinus arhythmia, heart block, auricular fibrillation, thyrotoxicosis, aortic stenosis, aortic insufficiency, tricuspid insufficiency, and coronary disease. It seems that this device should be valuable as an aid in teaching heart disease, since the difficulty presented by the fluoroscope, i.e., excessive exposure to patient and operator, is eliminated.

WILLIAM HARVEY SOCIETY

The William Harvey Society of the Tufts College Medical School held its first meeting of the new academic year in the amphitheatre of the Beth Israel Hospital on Friday, October 13. Dr. A. Warren Stearns, Dean of the Tufts College Medical School, introduced the speaker of the evening, Dr. William Gerry Morgan, formerly President of the American Medical Association. Dr. Morgan's subject was "Shakespeare's Knowledge of Medicine."

The speaker traced his interest in his subject back to a visit in England in 1912. Like his amazing grasp of many other fields of knowledge, Shakespeare's insight into things medical was far-reaching. There are no less than six hundred references to medical subjects in his thirty-seven plays. His views were, of course, the prevailing ones of the day. We find references to the influence of grief and peevishness in the production of jaundice. Many diseases are mentioned, ague (malaria) and the leanness associated with it, goitre, the "foul disease" or the "rotten disease of the South" (syphilis), and many others. That his knowledge was not superficial is shown by his use of terms not "lay" in any sense of the word, e.g., "tremor cordis," "hysterica passio," "pia mater". In speaking of the last, he mentions its relation to the brain, cord, and ventricles.

The medicine for soul and body ran hand in hand during the Elizabethan era. The clergy did much of the practice, but the barbers were the "surgeons" of the day and treated many of the bodily ailments with their favorite procedure of blood-letting. Fee-splitting was probably common even in those days. The pharmacopoeia consisted mainly of magic and so-called "shot-gun" prescriptions. Two hundred and eleven of the common prescriptions had more than ten ingredients, one had 230 components! The main influence was that of Hippocrates, somewhat modified by Galenism. Paracelsus' teaching was being felt.

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The next meeting of the William Harvey Society, of the Tufts College Medical School, will be held at 8 o'clock P.M. on November 10, in the auditorium of the Beth Israel Hospital, Boston.

The speaker will be Dr. Joseph Colt Bloodgood, Clinical Professor of Surgery, Johns Hopkins Medical School. His subject is "Irradiation in the Treatment of Cancer." The Chairman of the meeting will be Dr. Henry D. Chadwick, Commissioner of Public Health, Massachusetts.

Members of the medical profession are cordially invited to attend the meeting.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Amphitheatre (Van Dyke Street entrance), Tuesday evening, November 14, at 8:15 o'clock.

Présentation of Cases.

Subject: The Discovery of the Chemical Mediators of Autonomic Nerve Impulses. By Dr. Walter B. Cannon. JOHN HOMANS, M.D., *Secretary.*

HOUSE OFFICERS' ASSOCIATION OF THE BOSTON CITY HOSPITAL

TUESDAY, NOVEMBER 7, 1933

Subject—Syphilis

8:00 P.M.—Dr. John G. Downing

Primary and Secondary Syphilis
- Diagnosis
  - History
  - Dark Field
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- Symptoms and General Characteristics of Lesions
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Prophylaxis of Syphilis in General
- Importance of Early Diagnosis and Treatment
- Importance of Complete Examination of Patient
- General prognosis.

The Cutaneous Manifestations will be illustrated by lantern slides.

ROBERT T. PHILLIPS, M.D., *Secretary.*

THE TRUDEAU SOCIETY

There will be a meeting of the Trudeau Society on Wednesday, November 15, at 7 P.M. at the Essex County Sanatorium, Middleton, located on the Newburyport Turnpike.

The topic for the evening will be "Treatment of Pleural Effusions Complicating Artificial Pneumothorax Treatment." This will be presented by Dr. Olin S. Pettingill and his Staff.

MOSES J. STONE, M.D., *Secretary.*

GREATER BOSTON MEDICAL SOCIETY

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Speaker: Edgar Mayer, M.D., of Saranac Lake, New York.

Subject: "Cavities of the Lungs in Tuberculosis —Their Management."

Discussion: Edward D. Churchill, M.D.; Moses J. Stone, M.D.; Mark H. Joress, M.D.

Refreshments.

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November 19, December 17, January 15—Boston Medical History Club.

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ESSEX NORTH DISTRICT MEDICAL SOCIETY

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E. S. BAGNALL, M.D., Secretary-Treasurer.
281 Main Street, Groveland, Mass.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Thursday, November 2—Censors' Meeting at Salem Hospital, 3:30 P.M.

Wednesday, November 8—Salem Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. John Lovett Morse, Boston.

Wednesday, December 6—Beverly Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Hans Zinsser, Harvard Medical School. Subject: Bubonic Plague.

Wednesday, January 3—Danvers State Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Clarence A. Bonner, Superintendent of Danvers State Hospital. Subject: Recent Experiences in Foreign Clinics.

A very instructive paper on "X-Ray Features of Cranial Dysplasias of Pituitary Origin" was read by Dr. George Levene for Dr. Hector Mortimer, who was unavoidably absent. Changes in the functional state of the pituitary cause alterations of the skeleton, especially of the skull. Dr. Mortimer notes four main types, each of which has characteristic x-ray appearances.

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Wednesday, November 8—Salem Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. John Lovett Morse, Boston.

Wednesday, December 6—Beverly Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Hans Zinsser, Harvard Medical School. Subject: Bubonic Plague.

Wednesday, January 3—Danvers State Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Clarence A. Bonner, Superintendent of Danvers State Hospital. Subject: Recent Experiences in Foreign Clinics.

February 7—Council Meeting, Boston.

Wednesday, March 7—Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Frank H. Lahey, Boston. Subject: to be announced. Film Electrocardiogram.

Wednesday, April 4—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: to be announced. Subject to be announced.

Thursday, May 3—Censors' Meeting, at Salem Hospital, 3:30 P.M.

Tuesday, May 8—Annual Meeting. Salem Country Club, Forrest Street, Peabody. Dinner at 7. Speaker to be announced. Subject to be announced.

RALPH E. STONE, M.D., Secretary.

221 Cabot Street, Beverly, Mass.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of November, January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.

Sunderland, Mass.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Schedule 1933-1934

Meetings will take place on November 8 at the Bear Hill Club, Stoneham; January 10 at Melrose; March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, M.D., Secretary.

76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meetings will be held on January 31, and April 25 (Annual Meeting).

T. A. STAMAS, M.D., Secretary.

226 Central Street, Lowell, Mass.

NORFOLK DISTRICT MEDICAL SOCIETY

November 28—Roxbury Masonic Temple, 8 P.M. Pneumonia Symposium, in charge of Dr. Frederick T. Lord.

January 28—Tentatively Roxbury Masonic Temple, 8 P.M. Continuance of Symposium on Diseases of the Blood. Dr. Dameshek and Dr. Straus.

February 27—Place, time, subject, and speaker to be announced.

March 27—Place and time to be announced. Dr. Edward A. Edwards and Dr. Henry H. Faxon. Symposium on the Treatment of Varicose Veins.

May—Annual Meeting. Place, time and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.

1695 Beacon Street, Brookline, Mass.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

November 2—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Timothy Leary. Subject: Comparative Experimental Arteriosclerosis with Human Coronary Sclerosis.

December 7—12 noon at Quincy City Hospital. Program by the hospital staff.

January 4—12 noon at Norfolk County Hospital. Speaker to be announced.

February 1—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Sara Jordan. Subject: Stomach Ulcers.

March 1—12 noon at Quincy City Hospital. Program by the hospital staff.

April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.

Norfolk County Hospital, South Braintree, Mass.

SUFFOLK DISTRICT MEDICAL SOCIETY

November 2—Censors' Meeting will be held at the Boston Medical Library, 8 Fenway, at 4 o'clock.

November 29—Clinical Program at the Beth Israel Hospital.

January 31—General Meeting in association with the Boston Medical Library and the Trudeau Society. Speaker: Dr. Lawrason Brown of Saranac Lake.

March 28—Clinical Meeting at the Massachusetts Memorial Hospitals.

April 25—Annual Meeting at the Boston Medical Library. Election of Officers. Scientific Program and titles and speakers to be announced.

The Medical Profession is cordially invited to attend all of these meetings.

JAMES H. MEANS, M.D., Vice-President.
GEORGE P. REYNOLDS, M.D., Secretary.
311 Beacon Street, Boston, Mass.
ALEXANDER S. BEGG, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:

November 8—Dinner and scientific program at the Grafton State Hospital, North Grafton, Mass.

December 13—Dinner and scientific program at St. Vincent Hospital, Worcester, Mass.

January 10—Dinner and scientific program at the Worcester City Hospital, Worcester, Mass.

February 14—Dinner and scientific program at the Worcester State Hospital, Worcester, Mass.

March 14—Dinner and scientific program at the Memorial Hospital, Worcester, Mass.

April 11—Open date.

May 9—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.

27 Elm Street, Worcester, Mass.

BOOKS RECEIVED FOR REVIEW

1933 Year Book of Radiology. Diagnosis edited by Charles A. Waters, and Therapeutics edited by Ira I. Kaplan. Published by The Year Book Publishers, Inc. 804 Pages.

A Study of Rural Public Health Service. Allen W. Freeman, M.D., Editor. Published by The Commonwealth Fund. 236 Pages. Price $2.50.

The Treatment of Rheumatism in General Practice by W. S. C. Copeman. Published by William Wood & Company. 215 Pages. Price $3.25.

Manual of Urology by R. M. LeComte. Published by William Wood & Company. 317 Pages. Price $4.00.

A Practical Medical Dictionary by Thomas Lathrop Stedman. Twelfth Edition. Published by William Wood & Company. 1256 Pages. Price $7.50.

Behind the Doctor by Logan Clendening. Published by Alfred A. Knopf. 458 Pages. Price $3.75.

The Enlarged Prostate and Prostatic Obstruction by Kenneth M. Walker. Second Edition. Published by Oxford University Press. 223 Pages.

Bone Growth in Health and Disease by H. A. Harris. Published by Oxford University Press. 248 Pages.

Clinical Science Incorporating Heart. Thomas Lewis, Editor. Published by Shaw & Sons, Ltd. 158 Pages.

Surgical Anatomy by Grant Massie. Published by Lea & Febiger. 458 Pages. Price $6.00.

Infections of the Hand by Allen B. Kanavel. Published by Lea & Febiger. 552 Pages. Price $6.00.

Obstetrics and Gynecology by Arthur Hale Curtis, Editor. Published by W. B. Saunders Company. 1201 Pages.